Vol. III No. 4

HUMAN BIOLOGY

a record of research

DECEMBER, 1931

THE STRUCTURE OF A GROWING POPULATION

BY ALFRED J. LOTKA

Statistical Bureau, Metropolitan Life Insurance Company[1]

INTRODUCTION

For the sake of brevity, and to avoid technical terms, I have chosen for my title "The Structure of a Growing Population." I should state at the outset, however, that I propose to deal expressly with the characteristics of a population growing according to the logistic law.

As is well known, the logistic curve of growth has been found to fit certain populations rather well. One of the best, and, for us at any rate, the most important example of this, is the population of the United States, which, at the recent census of 1930[2] fell within 400,000, or less than one third of one per cent, of the calculated figure. I may say that we shall not here be concerned with a forecast, and therefore the question whether the population of the United States will continue to grow according to the logistic does not arise. We are here concerned only with the indisputable fact that it has closely followed the logistic in the past.

Now, a population which has for an extended period of time been following the logistic law has a definite pattern. Its demographic characteristics are not fortuitous or capricious, but conform to a type, or model, determined by the logistic law of growth itself. It is my purpose on this occasion to present a view of some of these typical characteristics of a logistic population and to show how the model compares

[1] Paper presented before the Second General Assembly of the International Union for the Scientific Investigation of Population Problems, London, July 18, 1931.

[2] Pearl, R., *Science*, 1930, Vol. 72, p. 399. The forecast of the population of the United States for 1930 by the logistic curve of Pearl and Reed, was 122.4 millions. The actual enumeration at the census was 122.8 millions.

with the actually observed population. We want to know, for example, what is the typical trend, in such a population, of the birthrate, the net fertility, the deathrate, the distribution by age and sex, the discrepancy between the "true" or "inherent" rate of natural increase and the observed excess of the birthrate over the deathrate.

In the treatment of the problem the following plan will be followed: We shall first of all examine the situation as it presents itself in a simplified or conventionalized case, namely, that of a population in which the life table remains unchanged and in which immigration and emigration are negligible.

Upon the results obtained for this simplified case we shall then build a representation of the more general case, corresponding to actual conditions, in which the life table is subject to a secular drift, and in which immigration and emigration form a notable part of the changes in the number of the population.

ARGUMENT

Consider the process by which the existing age distribution has come into being. Persons who are now ten years old were born ten years ago. Of the persons born at that time a certain fraction, determined by the life table, survives to the present day and forms the ten-year-old contingent of the existing population. A similar statement applies to every individual year of life.

Fundamental Equation: Relation Between Number of Population, Annual Births, and Life Table. Our fundamental formula, therefore, is

$$N(t) = \int_0^\infty B\ (t-a)\ p(a)da \qquad (1)$$

This states that the total number $N(t)$ of persons living at time t is the sum of the survivors to every age a of $B(t-a)$ persons born at time $t-a$ in a small interval of time da. The symbol $p(a)$ denotes the probability, at birth, of living to age a, that is, the fraction, out of a given large number born, that survive to age a.[3] The formula applies to an "isolated" population, in which immigration and emigration are negligible.

[3] In actuarial notation $p(a)$ would be written l_a/l_0.

Application to Logistic Population: Annual Births.[4] If we apply this fundamental equation to the case of a population growing according to the logistic law we have

$$\int_0^\infty B(t-a)p(a)da = \frac{N_\infty}{1+e^{-rt}} = N_\infty\,(e^{rt} - e^{2rt} + e^{3rt} - \ldots) \tag{2}$$

where N_∞ is the ultimate population.

Making the trial substitution

$$B(t) = U_1 e^{rt} - U_2 e^{2rt} + \ldots \tag{3}$$

we find that this satisfies the fundamental equation provided that

$$U_k = \frac{N_\infty}{\int_0^\infty e^{-kra}p(a)da} \tag{4}$$

We will write

$$b_k = \frac{1}{\int_0^\infty e^{-kra}p(a)da} = c_0 + c_1 kr + c_2 k^2 r^2 + \ldots \tag{5}$$

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[4] A preliminary and partial account of the development set forth in this section has been given by the writer in the *Proceedings National Academy of Science*, 1929, vol. 15, pp. 793-798. An analysis of a population in which the annual births (not the population) follow a logistic curve has been given by E. Zwinggi: Beiträge zu einer Theorie des Bevölkerungswachstums, 1929.

The problem here solved has been referred to by U. Yule (*Journal of the Royal Statistical Society*, 1925, vol. 88, Part I. page 35) in these words: "I see no general method of effectively attacking this problem at present and have contented myself with working out a couple of cases as illustrations." Yule sets up an initial population with an arbitrarily given age distribution, (namely, in one example a life table distribution, in his other example a Malthusian age distribution) and then computes step by step the ensuing age distribution and deathrates, hence birthrates.

It is to be noted that in the development set forth in the present communication no hypothesis of any kind is made regarding any initial age distribution. The problem is found to be entirely determinate without any such hypothesis, the logistic law defining its own age distribution for all time from $-\infty$ to $+\infty$. This therefore furnishes the answer to Yule's perplexity (*loc. cit.*, p. 55): "I was at first puzzled how to arrive at the population at $t=-4$ (of Yule's units) . . . without a good deal of troublesome approximation." Yule then cuts the Gordian Knot by assuming initially a Malthusian age distribution, a step which is pragmatically justified by the fact that logistic growth in its early stages is very nearly Malthusian. But in the method here set forth the perplexity of an initial age distribution does not arise at all.

The coefficients c are readily found by expanding the exponential function under the integral sign; denoting by L_n the nth moment of the function $p(a)$, that is, putting

$$\int_0^\infty a^n p(a)\,da = L_n \tag{6}$$

we find

$$\begin{aligned}
c_0 &= \frac{1}{L_0};\ c_1 = \frac{L_1}{L_0^2};\ c_2 = \frac{L_1^2}{L_0^3} - \frac{L_2}{2L_0^2};\\
c_3 &= \frac{L_1^3}{L_0^4} - \frac{L_1L_2}{L_0^3} + \frac{L_3}{6L_0^2};\\
c_4 &= \frac{L_1^4}{L_0^5} - \frac{3L_1^2L_2}{2L_0^4} + \frac{L_1L_3}{3L_0^3} + \frac{L_2^2}{4L_0^3} - \frac{L_4}{24L_0^2};\\
c_5 &= \frac{L_1^5}{L_0^6} - \frac{2L_1^3L_2}{L_0^5} + \frac{L_1^2L_3}{2L_0^4} + \frac{3L_1L_2^2}{4L_0^4}\\
&\quad - \frac{L_2L_3}{6L_0^3} - \frac{L_1L_4}{12L_0^3} + \frac{L_5}{120L_0^2},
\end{aligned} \tag{7}$$

Substituting these expressions for b_k in (3), and rearranging terms by collecting according to ascending powers of r instead of ascending powers of e^{rt}, we find

$$\begin{aligned}
B(t) = N_\infty\, e^{rt} \Bigg\{ &\frac{c_0}{(1+e^{rt})} + \frac{c_1 r}{(1+e^{rt})^2} + \frac{c_2 r^2(1-e^{rt})}{(1+e^{rt})^3}\\
&+ \frac{c_3 r^3(1-4e^{rt}+e^{2rt})}{(1+e^{rt})^4} + \frac{c_4 r^4(1-11e^{rt}+11e^{2rt}-e^{3rt})}{(1+e^{rt})^5}\\
&+ \frac{c_5 r^5(1-26e^{rt}+66e^{2rt}-26e^{3rt}+e^{4rt})}{(1+e^{rt})^6} + \dots\\
&+ \frac{c_j r^j}{(1+e^{rt})^{j+1}} \sum_{n=1}^{n=j} (-e^{rt})^{n-1} \sum_{\omega=0}^{\omega=n-1} (-1)^\omega \binom{j+1}{\omega} (n-\omega)^j + \dots \Bigg\}
\end{aligned} \tag{8}$$

$$= N_\infty \left\{ c_0 e^{rt} f(t) - \sum_1^\infty c_j f^j(t) \right\} \tag{9}$$

$$\text{where } f(t) = \frac{1}{1+e^{rt}} = \frac{1}{2} - \frac{1}{2} \tanh \frac{rt}{2} \tag{10}$$

and $f^j(t)$ denotes the jth derivative of $f(t)$ with respect to t. Or, writing $\phi(t) = \frac{1}{1+e^{-rt}} = \frac{1}{2} + \frac{1}{2} \tanh \frac{rt}{2}$ and noting that then $f^n(t) = (-1)^n \phi^n(t)$ we effect a slight simplification, namely,

$$B(t) = N_\infty \left[c_0\phi(t) - \sum_1^\infty (-1)^j c_j \phi^j(t)\right] \tag{11}$$

Computation is greatly facilitated by expressing $f(t)$ and $\phi(t)$ in hyperbolic tangent form.

Transformation of Origin.—The expression (8) for $B(t)$ can be simplified by the introduction of a new set of parameters b' and a shift of the origin of t, the time scale.

Transformation of coefficients c.—In analogy with the constants b_k defined above (5), let us introduce a new set of constants

$$b'_k = \frac{1}{\int_0^\infty e^{-kr(a-\tau)} p(a)\,da} \tag{12}$$

$$= \frac{1}{e^{kr\tau} \int_0^\infty e^{-kra} p(a)\,da} \tag{13}$$

$$= e^{-kr\tau} b_k \tag{14}$$

Now it is evident from (5) that

$$c_j = \left(\frac{1}{j!} \frac{d^j b_1}{dr^j}\right)_{r=0} \tag{15}$$

and similarly, in terms of the primed parameters

$$c'_j = \left(\frac{1}{j!} \frac{d^j b'_1}{dr^j}\right)_{r=0} \tag{16}$$

Thus, for example,

$$c'_1 = \left(\frac{db'_1}{dr}\right)_{r=0} = \left(-\tau e^{-r\tau} b_1 + e^{-r\tau} \frac{db_1}{dr}\right)_{r=0} \tag{17}$$

$$= -\tau b_0 + c_1 = c_1 - \tau c_0 \tag{18}$$

and similarly for higher coefficients. We thus find

$$
\begin{aligned}
c'_1 &= c_1 - \tau c_0 \\
c'_2 &= c_2 - \tau c_1 + \frac{\tau^2}{2!} c_0 \\
c'_3 &= c_3 - \tau c_2 + \frac{\tau^2}{2!} c_1 - \frac{\tau^3}{3!} c_0 \\
c'_4 &= c_4 - \tau c_3 + \frac{\tau^2}{2!} c_2 - \frac{\tau^3}{3!} c_1 + \frac{\tau^4}{4!} c_0 \\
c'_5 &= c_5 - \tau c_4 + \frac{\tau^2}{2!} c_3 - \frac{\tau^3}{3!} c_2 + \frac{\tau^4}{4!} c_1 - \frac{\tau^5}{5!} c_0
\end{aligned}
\tag{19}
$$

or in general

$$
c'_n = \sum_{k=0}^{k=n} \frac{(-\tau)^{(n-k)}}{(n-k)!} c_k \tag{20}
$$

These formulae apply quite generally for any value of τ. If, in particular, τ is the mean λ_1 defined by

$$
\lambda_1 = \frac{\int_0^\infty a\, p(a)\, da}{\int_0^\infty p(a)\, da} = \frac{L_1}{L_0}, \tag{21}
$$

then the formulae (19) reduce to very simple functions of the Thiele seminvariants of the function $p(a)$, as follows

$$
\begin{aligned}
c''_0 &= \frac{1}{L_0} \\
c''_1 &= 0 \\
c''_2 &= -\frac{\lambda_2}{2!L_0} \\
c''_3 &= \frac{\lambda_3}{3!L_0} \\
c''_4 &= -\frac{1}{4!L_0} \left\{ \lambda_4 - 3\lambda_2^2 \right\} \\
c''_5 &= \frac{1}{5!L_0} \left\{ \lambda_5 - 9\lambda_2\lambda_3 \right\}
\end{aligned}
\tag{22}
$$

Shifting of Origin of Time.—Of particular interest is a simultaneous shifting of the origin of time to $t—\lambda_1$, and introduction of the

parameters b', with $\tau = \lambda_1$. Let us consider how this will affect our fundamental equation. The fundamental equation (1) can indifferently be written

$$\int_0^\infty B[(t-\tau)-(a-\tau)]\ p(a)da = N(t) \tag{23}$$

$$= N_\infty\ [e^{rt} - e^{2rt} + \ldots] \tag{24}$$

Make the trial substitution

$$B(t) = U_1\ e^{\ r(t+\tau)} - U_2 e^{2r(t+\tau)} + \ \ldots \tag{25}$$

This gives

$$U_1 e^{rt} \int_0^\infty e^{-r(a-\tau)} p(a)da - U_2 e^{2rt} \int_0^\infty e^{-2r(a-\tau)} p(a)da + \ \ldots$$

$$= N_\infty\ (e^{rt} - e^{2rt} + \ldots) \tag{26}$$

so that the trial is effective provided that

$$U_k = \frac{N_\infty}{\int_0^\infty e^{-kr(a-\tau)} p(a)\,da} \tag{27}$$

$$= N_\infty\ b'_k \tag{28}$$

It is clear from this, that all the formulae developed with reference to the origin of time and age at zero, are directly applicable when referred to the origin of time $-\tau$, provided that we understand the coefficients c to be those defined above as c', and that the symbol t in the right hand member of (8) is understood to denote t', the time reckoned from $-\tau$ as origin, remembering that primarily the origin of time was taken at the center of the logistic curve $N(t)$.

In particular, when shift of origin is made to time $t - \lambda_1$, the formula for $B(t)$ becomes especially simple in view of the formulae (22) for the coefficients c''. We then have

$$B(t) = \frac{N_\infty}{L_0} \left\{ \varphi(t') + \frac{\lambda_2}{2!}\,\varphi''(t') + \frac{\lambda_3}{3!}\,\varphi'''(t') \right.$$

$$\left. + \frac{\lambda_4 - 3\lambda_2^2}{4!}\,\varphi^{IV}(t') + \frac{\lambda_5 - 9\lambda_2\lambda_3}{5!}\,\varphi^{V}(t') + \ \ldots \right\} \tag{29}$$

where $t' = t + \lambda_1$.

For purposes of computation it will be convenient to give this formula *in extenso*, as it appears when $\phi(t')$ is expressed in terms of the hyperbolic tangent:

$$B(t) = \frac{N_\infty}{L_0}\left\{\left(\frac{1}{2}+\frac{1}{2}\tanh\frac{rt'}{2}\right)+\frac{\lambda_2 r^2}{2!4}\left(\tanh\frac{rt'}{2}-\tanh^3\frac{rt'}{2}\right)\right.$$
$$-\frac{\lambda_3 r^3}{3!8}\left(1-4\tanh^2\frac{rt'}{2}+3\tanh^4\frac{rt'}{2}\right)$$
$$-\frac{(\lambda_4-3\lambda_2^2)r^4}{4!4}\left(2\tanh\frac{rt'}{2}-5\tanh^3\frac{rt'}{2}+3\tanh^5\frac{rt'}{2}\right)$$
$$+\frac{(\lambda_5-9\lambda_2\lambda_3)r^5}{5!8}\left(2-17\tanh^2\frac{rt'}{2}+30\tanh^4\frac{rt'}{2}-15\tanh^6\frac{rt'}{2}\right)$$
$$\left.-\ldots\right\} \qquad (30)$$

Numerical Example: The computation has been carried out on the basis of the United States life table 1919/20 (white females)[5] and the logistic curve of growth of the American population. The resulting curve of computed annual births is shown as a fully drawn line in the first panel of Figure 1. The components corresponding to successive terms in the series (30) are shown in the first and second panels of Figure 1.

It will be seen that the fundamental component

$$\frac{N_\infty}{L_0}\left(\frac{1}{2}+\frac{1}{2}\tanh\frac{rt'}{2}\right) \qquad (31)$$

accounts alone for nearly the entire contingent of births. The higher terms would hardly appear at all on the scale of the first panel in Figure 1 and have therefore been plotted separately on a scale twenty-five times greater in the second panel. A feature of special interest is that the third and the fifth term, whose presence disturbs the skew-symmetry of the resultant curve, are relatively very small; in consequence the resultant curve is very nearly skew-symmetric, resembling the simple logistic.

The curve takes its course between two asymptotes, the lower corresponding to a vanishingly small population and number of births,[6] the upper corresponding to the ultimate population N_∞ and a stationary

[5] Since we are dealing with a model population it is, in principle, immaterial what life table we use. Nevertheless it is desirable to use one corresponding as far as possible to the concrete existing conditions; see also footnote 8.

[6] Although population and births vanish for $t=-\infty$, the birthrate per head has a definite value, namely .0419.

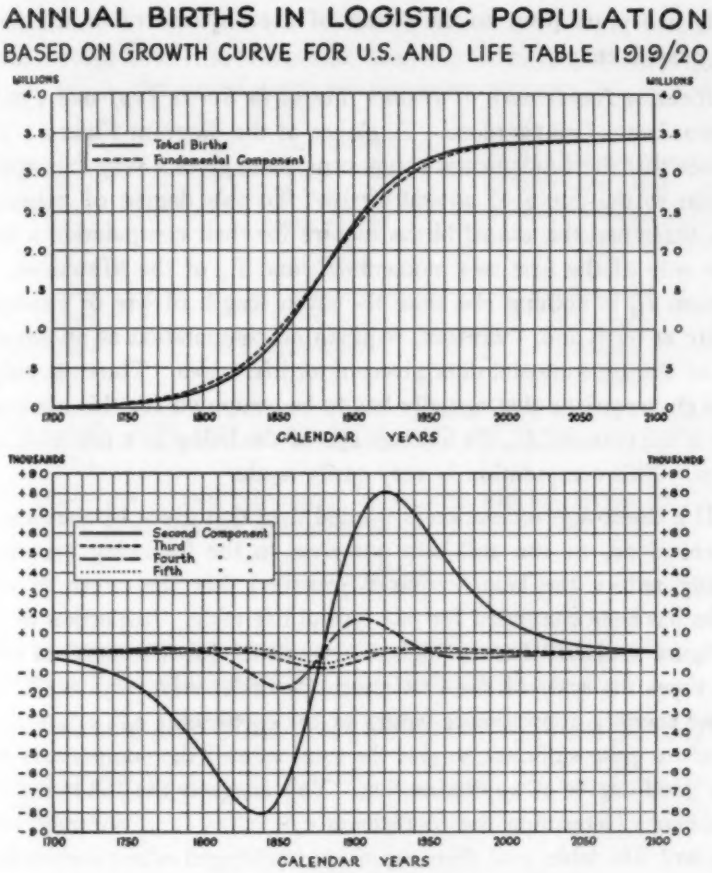

FIG. 1

birthrate equal to the reciprocal of the mean length of life. A point of special interest is that the half-way point of the curve of annual births is reached earlier than the half-way point of the population growth by a period very nearly equal to λ_1, the "mean age of the living in the life table population." This follows from the fact that the odd components [7] of the series (29) are very small (as already mentioned) and that the even components have, by the shift of origin, all been made to have their center of skew-symmetry at the point $t = -\lambda_1$, i. e., at a date

[7] *i. e.*, components containing odd derivatives of ϕ.

thirty-five years prior to the center of skew-symmetry of the population growth curve.

Relative Importance of Higher Terms in Series (29) and (30) and Approximate Computations.—A glance at the diagram Figure 1 shows at once that the fundamental component alone gives a very fair approximation to the curve of annual births. To this degree of approximation, therefore, the annual births require for their computation a knowledge only of the first two moments L_0 and L_1 of the life curve. The moment L_0 is nothing else than the mean length of life or expectation of life at birth and, therefore, requires no computation at all since it is one of the fundamental data given in all life tables. Thus the only life table characteristic that actually has to be computed for this approximation is the moment L_1, the average age of the living in a life table population. This computation is very easily made.

If a closer approximation is wanted than that given by the first component alone, we can still help ourselves in the following manner and greatly reduce the labor required provided that the curve of annual births has been computed for one typical life table. Inspection of Panel 2, Figure 1, shows that the higher components (shown there on twenty-five times the scale of the first panel) are relatively very small. Even rather roughly approximate values of the coefficients c_2'', c_3'', . . . are therefore quite sufficient to give the total sum of the components with a very good degree of approximation. This suggests the following simple expedient: Determine the coefficients c_2'', c_3'', . . . for any suitable standard life table, and then use these unchanged when computing the annual births on the basis of any other life table. It goes without saying that it is desirable to use life tables not too widely divergent, but the latitude permissible is very considerable.

Numerical Example.—The following example shows with what very satisfactory degree of accuracy it is permissible to use coefficients c_2, c_3 . . . . from a previously employed life table, in computing the annual births (in a logistic population) on the basis of a new life table, thus saving the labor of computing new values of these coefficients, only the moments L_0 and L_1 of the new life table being actually required.

TABLE 1

Annual Births in Logistic Population, Computed on Basis of Growth Curve for United States and Life Table 1919-1920

YEAR	ALL COEFFICIENTS, c_k, ACCURATELY DETERMINED FOR 1919-20 LIFE TABLE	ONLY MOMENTS L_0 AND L_1 ACCURATELY DETERMINED FOR 1919-20 LIFE TABLE. HIGHER COEFFICIENTS, $c_2, c_3 \ldots$, TAKEN FROM 1910 LIFE TABLE
1800	218,695	218,654
1850	890,975	890,880
1900	2,330,669	2,330,740
1950	3,164,261	3,164,439
2000	3,373,610	3,373,512

NOTE: The reader should be reminded that we are dealing with a model population having the same logistic growth curve as the United States, but growing entirely by excess of births over deaths (that is, without immigration) and with a fixed life table.

Birthrate per Head.—Having found the annual births $B(t)$, the birthrate per head follows immediately as

$$b(t) = \frac{B(t)}{N(t)} \tag{32}$$

It will be seen from the graph of $b(t)$ in Figure 2 that this is a nearly skew-symmetric curve resembling an inverted logistic.

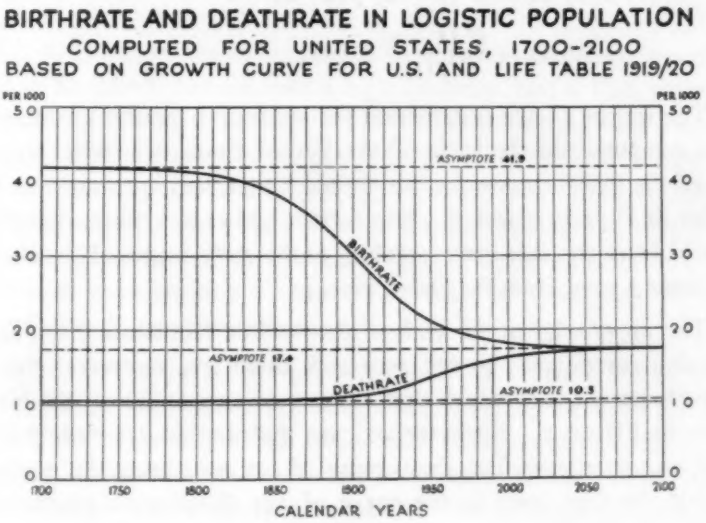

FIG. 2

Inspection of the curve (Fig. 2) shows that in the early stages of the development of a logistic population the birthrate declines at first very slowly, then more rapidly. Then follows a period of rather rapid decline, giving way finally to a period of slow decline towards a final stationary level. The point of mid-level is, in this case, reached in the year 1902 about twenty-two years later than the mid-level of the total births (1880) but about twelve years before the mid-level of the population (1914).

Deathrate.—The birthrate having been determined, and the rate of increase being given by the logistic curve of growth, the deathrate immediately follows by subtraction as rate of increase minus birthrate. The figures thus computed for the case of fixed life tables are represented graphically in Figure 2 together with the birthrate curve. The detailed discussion of this curve may with advantage be deferred to a later section, where comparison will be made with the corresponding curve as computed on the basis of a fluent life table.

Age Distribution.—Given the annual births and the survival function $p(a)$, the age distribution follows at once; for, the persons which at any particular moment are between a and $a + da$ years old, are the survivors of those born at time $t - a$ in a time interval da, that is to say

$$N(t)\, c(a)\, da = B\,(t-a)\, p(a)\, da \tag{33}$$

$$c(a) = \frac{B\,(t-a)}{N(t)}\, p(a) \tag{34}$$

The curves of age distribution for successive quarter centuries have been computed on the basis of the United States life table 1919-1920 and of the logistic curve for the United States, and are shown as a solid model in Figures 3 and 4. The former presents a view of the model as seen from the face corresponding to the early stages of development, the latter a view from the "late" face.

The upper edge at the back of the surface representing the age distribution corresponds to age zero and, therefore, represents the curve of birthrate per head which thus appears as a repetition of the birthrate curve in Figure 2. Birthrate and age distribution are closely interrelated and interdependent, the persons of age zero being the newly born so that the first point in the curve of age distribution represents the birthrate per head.

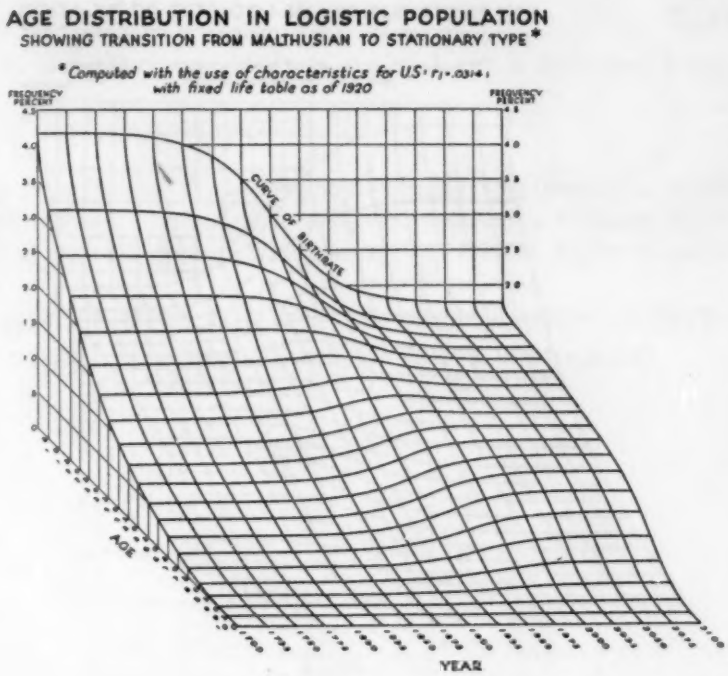
AGE DISTRIBUTION IN LOGISTIC POPULATION
SHOWING TRANSITION FROM MALTHUSIAN TO STATIONARY TYPE*
* Computed with the use of characteristics for U.S. r1=.0314 with fixed life table as of 1920
FREQUENCY PERCENT
CURVE OF BIRTHRATE
AGE
YEAR

FIG. 3

In the early stages the curve of age-distribution is concave upward throughout its extent, and exhibits a high birthrate, and a high proportion of young persons. (See Figure 3.) In the late stages (see Figure 4) the age distribution is convex upward over a considerable range of ages; birthrate and proportion of young people are low. At intervening stages the age distribution passes through transitional types, giving rise to the "warped" surface of the diagram. It will be worth while to give a little closer consideration to these two extreme types and to the transitional type of age distribution.

Stable or Malthusian Age Distribution.—It will be remembered that in its early stages the logistic law of growth coincides nearly with the Malthusian law of geometric rate of increase (compound interest law). Accordingly, the age distribution in the early stages of the growth of a population is very nearly the Malthusian (or "stable") age distribution corresponding to the fertility and mortality which prevail at the time,

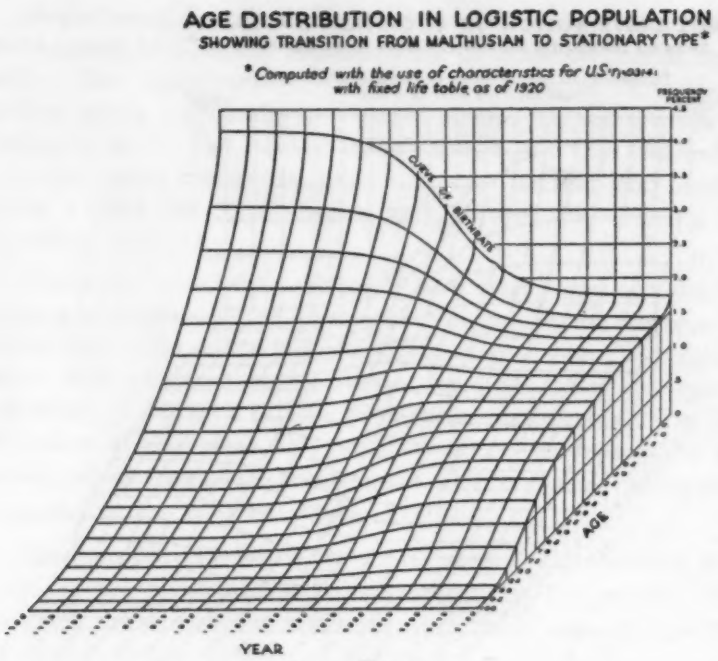
AGE DISTRIBUTION IN LOGISTIC POPULATION
SHOWING TRANSITION FROM MALTHUSIAN TO STATIONARY TYPE*
* Computed with the use of characteristics for U.S. 1910-1914 with fixed life table as of 1920
CURVE OF BIRTHRATE
AGE
YEAR

FIG. 4

and which at this stage change very slowly, that is, are nearly constant. The form of this Malthusian or stable age distribution is

$$c(a) = be^{-ra}p(a) \tag{35}$$

where $c(a)$ is the coefficient of age distribution, such that $c(a)da$ gives the proportion, in the total population, of persons whose age is comprised within the limits a and $a + da$; the symbol b denotes the birth-rate per head per annum; $p(a)$ is the probability,[8] at birth, of reaching age a, and r is the incipient rate of natural increase per head, which for the United States has a value of .0314 per head.

Stationary or Life Table Age Distribution.—In its terminal stages a population following the logistic curve of growth approaches a stationary state. The age distribution is then not only stable, but conforms directly to the life table, since the formula (35) above, when $r = 0$, reduces to

[8] Commonly denoted, in actuarial notations by l_a/l_0, or simply by l_a if it is understood that the radix of the life table is unity at age zero.

$$c(a) = b_0\, p(a) \tag{35a}$$

where b_0 is the stationary birthrate per head, and is equal to the reciprocal $\frac{1}{L_0}$ of the mean length of life.

Age Distribution: Plane Diagram.—Another representation of the trend of the age distribution in a logistic population is shown in the "plane" diagram Figure 5, which brings out certain important points

AGE DISTRIBUTION FOR LOGISTIC POPULATION GROWTH
COMPUTED FOR THE UNITED STATES, 1850-2000
ON BASIS OF LIFE TABLE 1919-1920

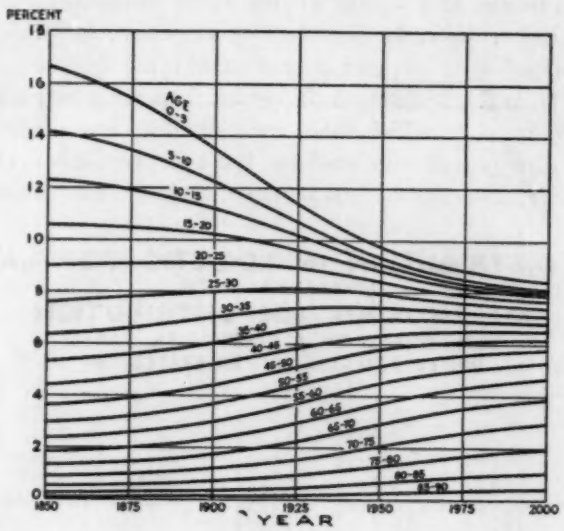

FIG. 5

with particular clarity. As regards the general character of the diagram it is to be noted first of all, that for the younger ages the trend of the proportion of persons of a given age is systematically downward, in the form of an inverted S. When we come to the age group 20 to 25, we have a transitional type of curve which begins with a slight upward gradient and then ends in an inverted S. For the higher age groups, the upward gradient at the beginning becomes more and more marked and at the same time the inverted S becomes transformed into an S in its

natural position, so that the general trend for the curves of the higher ages is upwards. This is quite characteristic—the lower age groups diminish proportionally and the higher age groups increase, so that the population, on the whole, becomes older. If we look at that part of the diagram which corresponds about to the present date, 1930, we observe the following facts. Age groups 0 to 5, 5 to 10, 10 to 15, 15 to 20, 20 to 25 are on a downward grade. Age group 25 to 30 is just at the turning point from an upward to a downward grade. Essentially the same is true of the next age group 30 to 35, while higher age groups are on an upgrade and will remain so until they flatten to a horizontal when a stationary population is reached.

Comparison of Malthusian with Logistic Age Distribution.—While the age distribution in a logistic population is practically identical with the Malthusian in the early period of development, in its characteristic form corresponding to the transitional period, the logistic age distribution differs from the Malthusian in certain important respects. This is shown in Figure 6, in which there are plotted on one diagram, a.) the logistic age distribution computed for the 1920 population on the basis of a fixed life table (as of 1919-1920); and, b.) the Malthusian age

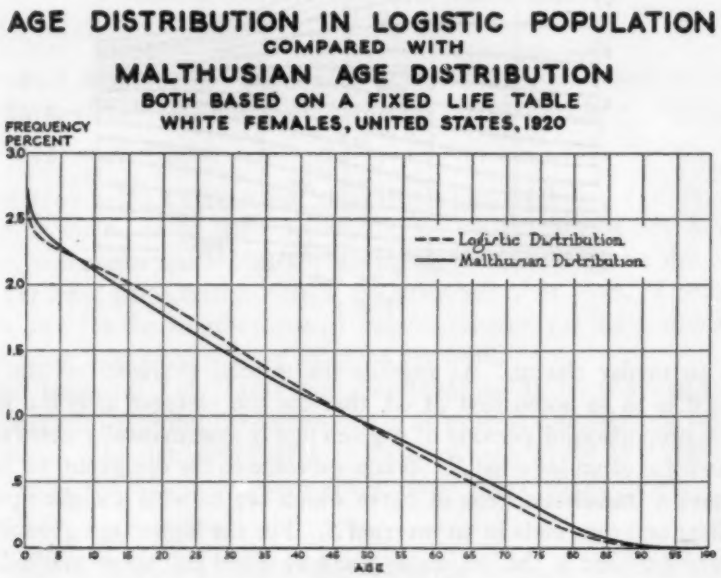

FIG. 6

distribution, computed on the basis of the same life tables but for a constant rate of increase per head $r_t = .014$, the value given by the logistic curve for the year 1920.[9]

The important point to note is that logistic age distribution rises above the Malthusian for early adult life (ages 10 to 45), but falls below it at ages above 45. It is this characteristic of the logistic age distribution that gives the population a natural rate of increase momentarily greater than that actually corresponding to existing fertility and mortality, as discussed in a later section below.

The Secular Drift in Fertility.—Since, in the preceding development the mortality (life table) of the population has been considered constant, the continually diminishing rate of increase per head r_t characteristic of the logistic law of growth is referrable to a diminishing fertility. We may, therefore, inquire what is the law of this diminishing fertility.

Now speaking quite generally, the fertility cannot be defined by a single figure, since it represents the resultant of age-specific fertilities integrated over the entire period of reproduction. A given rate of increase can therefore arise, with a given life table, from a variety of different fertility curves. To make the problem determinate, some restricting assumption must be made regarding the mode of variation of the fertility curve. The simplest assumption, and the only one which shall here be used as a basis of discussion, is that the curve of fertility as a function of age (for females)[10] changes in amplitude only, while keeping its shape otherwise unchanged. With this assumption it is possible to compute the relative fertility at any instant as compared with the given fertility (1920) standard. For, if we denote by $m(a, 1920)$ the maternity frequency (counting daughters only) at age a in the standard year, 1920, and by $m(a,t) = k(t)m(a,1920)$ the corresponding maternity frequency at time t, then we have evidently,

[9] Equation (35) gives the Malthusian age distribution for a special case, namely that in which the rate of natural increase has the incipient value $r = .0314$. A Malthusian age distribution can, of course, also be computed for any other rate of natural increase, such as r_t, the rate corresponding to the logistic at time t. See Appendix, equation (60).

[10] It is most convenient to carry out the entire computation on the basis of the female population, because the period of reproduction of females has a more definite and earlier termination than that of males. In the equation (36) the symbols $p(a)$ and $m(a)$ are therefore to be read as referring to the female population.

$$B(t) = k(t) \int_0^\infty B(t-a)\ p(a)\ m(a,1920)\ da \qquad (36)$$

This equation defines $k(t)$ in terms of quantities known or determined above.

The values of $k(t)$, the *relative fertility* (in terms of the standard fertility of 1920), computed for a succession of calendar years are set forth in Table 2 below together with the corresponding ratio $R(t)$ between the total births in two successive generations. This ratio $R(t)$ itself is a sort of measure of net fertility, and has the advantage that it automatically assumes the value unity for a stationary population. It is not, however, a complete measure of net fertility because it is expressed in terms of a vague unit of time, namely, the "mean length of a generation."

Having ascertained the fertility coefficient $k(t)$ as a function of the time, we can at once proceed to derive therefrom two other demographic characteristics of interest, namely, (1) the "true" or "inherent" rate of natural increase, and (2) the average size of families per wife, or, if the proportion of sterile marriages is known, per mother.

The True or Inherent Rate of Natural Increase: Comparison with Excess of Birthrate per Head Over Deathrate per Head.—It has been shown elsewhere [11] that the true or inherent rate of natural increase ρ_0 is given by the real root of the equation.

$$1 = \int_0^\infty e^{-\rho a}\ p(a)\ m(a)\,da \qquad (37)$$

where $m(a)$ is the maternity frequency per head of female population of age a, counting births of daughters only. It is to be noted that the inherent rate of increase so defined is a definite measure of the net fertility of the population, reckoned no longer in the vague unit of a generation, but in the common unit of time, i. e. per calendar year.

In the present case $m(a)$ is a function of the time, and, according to the assumption made in the preceding section we write

$$m(a,t) = k(t)\ m(a,1920) \qquad (38)$$

where $m(a)$ now refers specifically to the standard maternity frequency, i. e., in this case that of 1920. Thus we have

[11] *Phil. Mag.*, 1911, Vol. 21, p. 436; *Journal American Statistical Association*, 1925, Vol. 20, p. 329.

$$1 = k(t) \int_0^\infty e^{-\rho a}\, p(a)\, m\,(a,1920)\,da \tag{39}$$

Solving (39) explicitly for ρ, we find [12]

$$\mu_2 \frac{\rho^2}{2} - \mu_1 \rho + \log_e k(t)\, R_0 = 0 \tag{40}$$

$$\rho = \frac{\mu_1 - \sqrt{\mu_1^2 - 2\mu_2 \log_e k(t) R_0}}{\mu_2} \tag{41}$$

$$= F(t) \tag{42}$$

where

$$\mu_1 = \frac{R_1}{R_0} \tag{43}$$

$$\mu_2 = \frac{R_2}{R_0} - \left(\frac{R_1}{R_0}\right)^2 \tag{44}$$

and where R_k is the kth moment of the function $p(a)\, m(a,1920)$.

COMPARISON OF TRUE RATE OF NATURAL INCREASE
WITH
EXCESS OF BIRTHRATE OVER DEATHRATE
For Logistic Population Growth

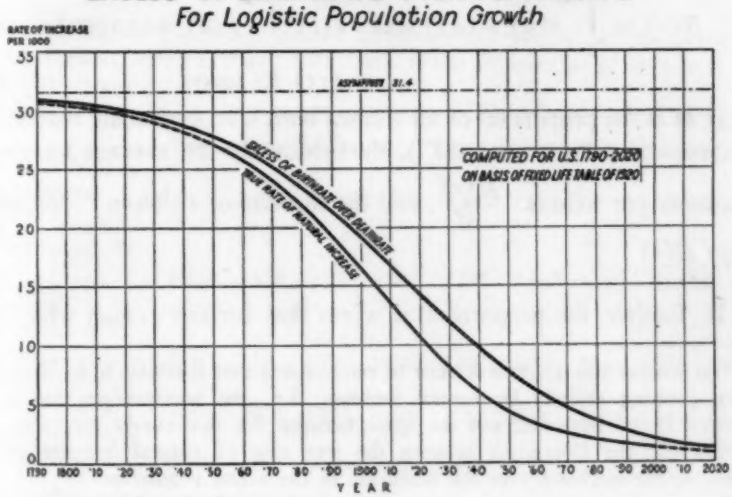

FIG. 7

[12] For the method of solution see *Journal of the American Statistical Association*, 1925, pp. 331-332. For values of ρ such as occur in practice terms of higher than second degree in ρ are negligible and have here been omitted.

Figure 7 shows the graph of the function $F(t)$, the "true rate," and side by side with it, the graph of the observed rate of natural increase, i. e. the excess of the birthrate over the deathrate. It will be noted that, throughout the history of a population growing according to the logistic law, the true or inherent rate of natural increase falls systematically below the excess of birthrate over the deathrate, but these two quantities approach each other more and more as we pass either to the extreme right or to the extreme left of the diagram. Thus in the early stages of the development of a population growing according to the logistic, where this law of growth is nearly identical with the compound interest law, the excess of birthrate over deathrate and the true rate of natural increase practically coincide; and they again practically coincide in the last stages, when the population is nearly stationary and the age distribution is nearly a life table distribution.

Throughout the intervening period the "true" or inherent rate of natural increase falls below the excess of the birthrate over the deathrate.[13]

Size of Families. Number of Children per Mother.—The ratio of total female births in two successive generations is evidently given by

$$R(t) = \int_0^\infty p(a)\, m(a,t)\, da = k(t) \int_0^\infty p(a)\, m(a,1920)\, da \quad (45)$$

$$= k(t)\, R(1920) \quad (46)$$

If M is the proportion of all women born who eventually marry (i. e. the proportion "ever married"), then evidently the average number of daughters per wife is $\frac{R(t)}{M}$, and the number of children[14] per *wife* is $\frac{2.057\, R(t)}{M}$.

If, further, the proportion of wives that for any reason whatever[15]

[13] In reading this diagram it must be remembered that it relates to an ideal population growing entirely by natural increase; i. e., the immigration rate is here included in the birthrate, and the space between the two curves represents only qualitatively the divergence between the true rate of natural increase and the excess of the birthrate over the deathrate in the actual population.

[14] In the United States the sex ratio at birth (male: female) has been fairly constant at 1.057 for years past.

[15] That is to say, either from inherent (true) sterility, or through premature disruption of marriage by death of wife or husband, or by divorce. *See* A. J. Lotka, *Proceedings of the National Academy of Science*, 1928, volume 14, p. 99.

bear no children ("gross" sterility) is s then the average number of children per *mother* is

$$C = \frac{2.057\, k(t)\, R(1920)}{(1-s)\, M} \qquad (47)$$

Numerical Data.—The sterility s and the proportion "ever married" have no doubt undergone considerable changes in the course of past decades and centuries. We are, however, at this stage considering a conventionalized model population with a fixed life table and growing entirely by natural increase, i. e. without immigration or emigration. For the present we will, therefore, merely substitute constant values for these coefficients, namely, the values prevailing in the United States in 1920: $s = 17.1$ per cent, $M = 78.2$ per cent. We then find for C, the number of children per mother, the figures set forth in Table 2. The actual figures for earlier calendar years must have deviated from those here shown as follows: (1) A deduction must be made on account of net immigration, the effect of which is here tacitly included in the births. (2) A further deduction must be made on account of a lower sterility in the past. (3) It is somewhat uncertain whether the correction for

TABLE 2

Net Fertility in Logistic Population Computed on Basis of Growth Curve for United States and Fixed Life Table, White Females, as of 1919-1920

	YEAR									
	1800	1825	1850	1875	1900	1920	1925	1950	1975	2000
Ratio, $k(t)$, of fertility to standard (1920)....	2.011	1.951	1.836	1.639	1.371	1.162	1.119	.971	.911	.886
Ratio, $R(t)$, of total births in two successive generations	2.329	2.260	2.127	1.899	1.588	1.346	1.296	1.125	1.056	1.027
Number of children per mother, (with 17.1% gross sterility).....	7.40	7.18	6.76	6.03	5.05	4.28	4.12	3.57	3.35	3.26
"True" or "Inherent" rate of natural increase, ρ_0..	.0304	.0293	.0271	.0229	.0165	.0105	.0092	.0042	.0019	.0009
Rate of increase according to logistic curve, r_t ...	.0306	.0296	.0277	.0243	.0191	.0143	.0131	.0077	.0040	.0020

NOTE: In studying the figures in the table it must be borne in mind that they refer to a model population, having the same total rate of increase per head as the logistic curve for the United States at the same date, but growing solely by excess of births over deaths. In other words the net immigration has been, as it were, included in the birthrate. So, for example, the reason why the figure 1.162 for $k(t)$ in the standard year (1920) is not unity, is that it refers to the *model* population in which population growth takes place wholly by natural increase; whereas the standard fertility is that actually observed in the *real* population in 1920.

Again, in reading the figures for dates later than 1925, it must be understood that these have been given merely in order to complete the model, *not by the way of a forecast.*

proportion of married should be positive or negative. (4) A positive correction would have to be made on account of the much less favorable life table at earlier dates. (5) There has undoubtedly occurred a change in the profile, as well as the amplitude, of the age-fertility curve.

It should be remarked that in the case of the year 1920, the standard at the basis of this computation, the second and third corrections enumerated above drop out, and the chief remaining correction is that for net immigration. As a matter of fact, a separate determination of the size of families for that year, by the direct method which does not involve immigration, gives 3.68 children per mother.[16]

The difference between this figure of 3.68 and the 4.3 shown in the table indicates at least approximately how much greater the average family would have had to be in 1920 in order to produce the actual rate of increase without the aid of immigration.

The Sex Ratio.—Just as the logistic population has a characteristic age distribution, so also the distribution according to sex is definite at each stage in the growth of the population. The ratio of males to females in the total population (all ages) is simply given by

$$\frac{N_m}{N_f} = \frac{B_m}{B_f} \cdot \frac{b_f}{b_m} \tag{48}$$

$$= 1.057 \frac{b_f}{b_m} \tag{49}$$

where the subscripts m and f denote that the quantities to which they are affixed refer respectively to the male and the female population considered separately.

For obvious reasons the sex ratio so computed varies within rather narrow limits. Thus computation gives the following sequence of figures (on the basis of the United States Life Tables for white males and females 1919-1920 and a sex ratio at birth of 1.057 males per one female).

The ratio actually observed in 1920 among the native white population was 1.0173. In view of the fact that the computation was carried out on the basis of fixed life tables and without regard to immigration, only a qualitative agreement with the observed figure could here be expected.

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[16] *Journal American Statistical Association*, 1925, p. 325.

TABLE 3

Sex Ratio in Logistic Population
Computed on Basis of Growth Curve for United States, and Fixed Life Table, as of 1919-1920

CALENDAR YEAR	RATIO OF MALES TO FEMALES IN WHITE POPULATION	CALENDAR YEAR	RATIO OF MALES TO FEMALES IN WHITE POPULATION
1800	1.0282	1920	1.0219
1850	1.0276	1950	1.0188
1900	1.0245	2000	1.0172

INFLUENCE OF CHANGING MORTALITY

So far the discussion has been conducted on the basis of a fixed life table, the reason for this being that this case admits of a direct and exact mathematical treatment for a logistic population. As was pointed out in the Introduction, we can now proceed, upon the results obtained for the simplified model population, to build a method for treating the more general case, corresponding to actual conditions, with a fluent life table, and with allowance for immigration. The procedure is best explained by the aid of the diagram Figure 8.

On this diagram will be seen a number of birthrate curves drawn in lines of dashes. Each of these was obtained by computation in pre-

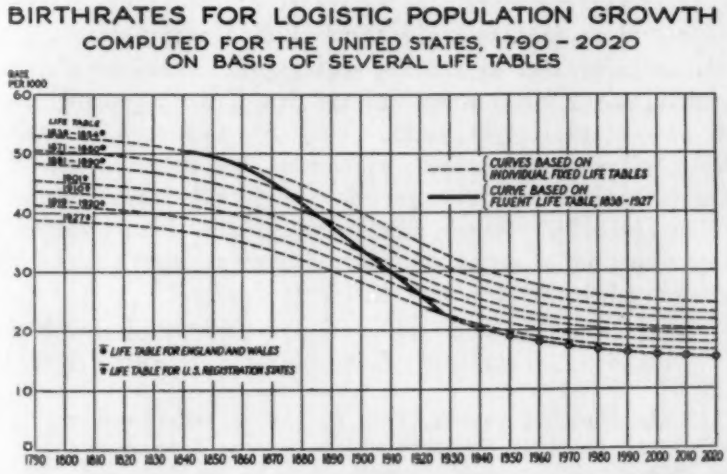
BIRTHRATES FOR LOGISTIC POPULATION GROWTH
COMPUTED FOR THE UNITED STATES, 1790 - 2020
ON BASIS OF SEVERAL LIFE TABLES
RATE PER 1000
60
50
40
30
20
10
0
CURVES BASED ON INDIVIDUAL FIXED LIFE TABLES
CURVE BASED ON FLUENT LIFE TABLE, 1830-1927
LIFE TABLE FOR ENGLAND AND WALES
LIFE TABLE FOR U.S. REGISTRATION STATES
1790 1800 1810 1820 1830 1840 1850 1860 1870 1880 1890 1900 1910 1920 1930 1940 1950 1960 1970 1980 1990 2000 2010 2020

FIG. 8

cisely the same manner as the curve appearing at the top of figures 3 and 4, except that a different life table was used for each of the dashed curves. The lowermost curve, corresponding to most recent conditions, is based on a life table for 1927, which was the latest available at the time this diagram was made. The next previous in order is based on the United States life table for 1919-1920, the last official life table published by the Census Bureau. Prior to these are shown the curves based on the life tables of 1910 and 1901. Earlier than this we have no American life table, and it was necessary to employ English life tables corresponding to the earlier dates. A reasonable justification for this is found in the fact that English life tables generally closely resemble ours, and in the further fact that the results as plotted were quite harmonious with those based on later life tables.[17]

On each of these inverted *S* shaped curves a ten year segment is drawn in a heavy line, this segment representing the ten years centered at the calendar year to which the life table primarily applies. When mortality is actually undergoing the secular changes corresponding to the successive life tables, the curve truly representing the relation between the birthrate and the time in calendar years must pass through these heavily drawn segments approximately at their midpoint. In fact, we may think of the true curve as being formed by each of these heavily drawn segments being turned around until they form a continuous curve; that continuous curve has been drawn through in a heavy line, and represents essentially the course of the birthrate under the conditions of changing mortality to which the diagram relates.

Remembering that we are here speaking of a model population, and not attempting a forecast, we can say that if the population were to continue to grow according to the logistic law, then the birthrate would have to follow a curve closely similar to that shown in dashes and circles at the right hand lower portion of Figure 8, which is drawn with the New Zealand life table in mind (expectation of life 65 years). The picture would not be very greatly altered if we assumed a slightly better mortality such as may in time be achieved.

It will be seen that, on the assumption of a continued growth according to the logistic law and with a fluent life table, the course of the birth-

[17] As pointed out in a previous section, the birthrate curve based on a fixed life table is almost completely determined by the first two moments of the life table alone. Minor differences in the general outline of the English and the United States life tables are, therefore, inconsequential.

rate is again represented by an inverted *S*-shaped curve, as it was when computed on the basis of a fixed life table, but now with a much steeper middle limb.

Deathrate.—It is interesting to compare the general character of the deathrate curve as computed for a fluent life table (Fig. 9) with that

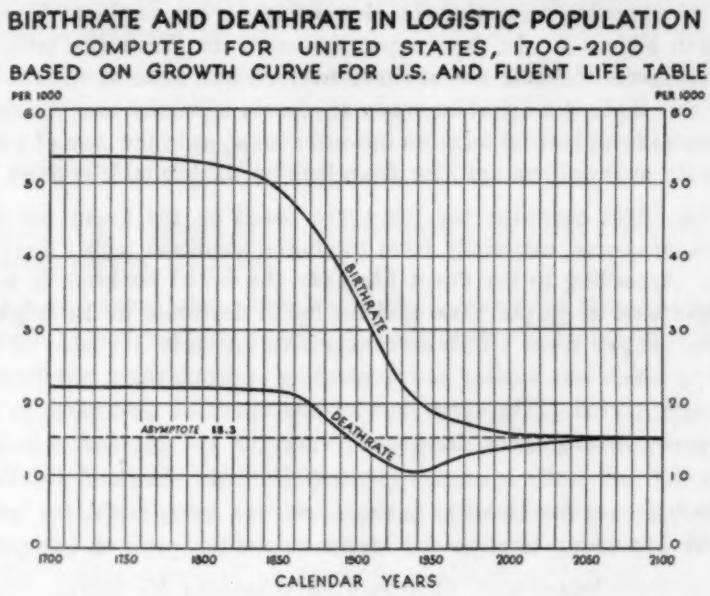

FIG. 9

as computed for a fixed life table (Fig. 2), as there is a very decided difference between the two. For a fixed life table the deathrate curve takes the form of a very flat *S*, starting from a lower asymptote of 10.5 per 1,000 per annum (as computed on the basis of the 1919-1920 life table), and turning upwards to seek finally the level of the upper asymptote at 17.4, where it joins the birthrate that approaches the same asymptote from above.

The curve computed on the basis of a fluent life table is quite different in character. After remaining nearly level for a considerable period, it presently turns downward following the general course of the birthrate until about the year 1930 or 1940, after which it turns up again and approaches the common asymptote on which birthrate and

deathrate unite to the level corresponding to a stationary population for the life table that has been selected as the ultimate life table.

A characteristic feature of this deathrate curve is a descending portion between the years 1850 to 1930. This is the result of the improving mortality which finds expression in the fluent life table, and which, of course, is not brought to expression when a fixed life table is used as the basis of computation, so that this descending portion is absent from the curve in Figure 2. In the computation with the fluent life table, the final optimum life table was assumed to come into force at 1950 so that after this there is no further improvement in mortality and the deathrate curve from here on takes an upward course, as in the case of a fixed life table, as is only natural, for from here on the life table *is* fixed.

The initial condition for the curve based on the fluent life table differs, of course, materially from the initial condition with a fixed life table. According to the fluent life table the initial birthrate is in the neighborhood of 53 per 1,000 and the initial deathrate in the neighborhood of 22 per 1,000. This corresponds to the early life table of 1838 to 1854 which was applied as representing approximately conditions at this early period. The early part of the curve of deathrates is very flat, partly because here the growth curve of the population is itself rather flat, and partly because a constant life table (England and Wales 1838-1854) was here used as basis, no attempt being made, for lack of suitable life tables, to allow for higher mortalities prior to this period.

Age Distribution with Allowance for Changing Mortality.—Having learned how to allow for the secular changes in mortality in computing the annual births, we can go back to the computation of the age distribution on the basis of a fluent life table and of the corresponding birthrates. This more exact computation has been carried out only for the age distribution in the year 1920.

Method of Using Fluent Life Table in Computing the Age Distribution.—Consider a block of persons a years old in 1920, for example. They were born in the year $1920 - a$. In the year of their birth they were subject to a mortality corresponding to a life table for the year $1920 - a$. We shall assume that this life table is computed with sufficient accuracy by linear interpolation between the two nearest actually available life tables between which $1920 - a$ lies. For example, if a is less than 10, we shall use a survival factor.

$$p(a,1920-a) = p(a,1910) + \frac{(1920-a)-1910}{1920-1910}\{p(a,1920)-p(a,1910)\} \tag{50}$$

and similarly for values of a for which the year of birth lies between any other two nearest available life tables.

In this way, for persons of each age a, a life table corresponding to the year of birth was used.[18]

With this method of establishing a fluent life table, we now employ a modification of the age distribution formula (34), namely

$$c(a,t) = \frac{B(t-a)}{N(t)}\ p(a,t-a) \tag{51}$$

where $B(t)$ is computed by the special method set forth above with use of a succession of life tables.

It is understood that there is a distinctly empirical element in these methods. Their justification is to be seen largely in the results, as will appear in the sequel.

Comparison of Various Types of Age Distribution.—In a previous paragraph and diagram (Figure 6) comparison was made between the age distribution in a logistic population, as computed on the basis of a *fixed* life table (1920), and the corresponding Malthusian age distribution. A similar comparison can now be made between the logistic age distribution computed on the basis of the *fluent* life table, and the Malthusian distribution. This comparison is shown in Figure 10. It will be noted that in general character this diagram closely resembles the one previously constructed on the basis of a fixed life table, but that the characteristic features pointed out in the former diagram reappear in accentuated form: there is an excess of persons below the age forty-five and a deficiency of persons above that age in the logistic, as compared with the Malthusian age distribution.

Another comparison of particular interest is that between the logistic age distribution, computed on the basis of the fluent life table, and the *actually observed* age distribution. This comparison is made in Figure

[18] An alternative and more refined procedure would be to apply to each age class a mortality (probability of dying within one year) $q(a,t)$ obtained by interpolation between the two nearest available life tables. The application of this method would be excessively laborious.

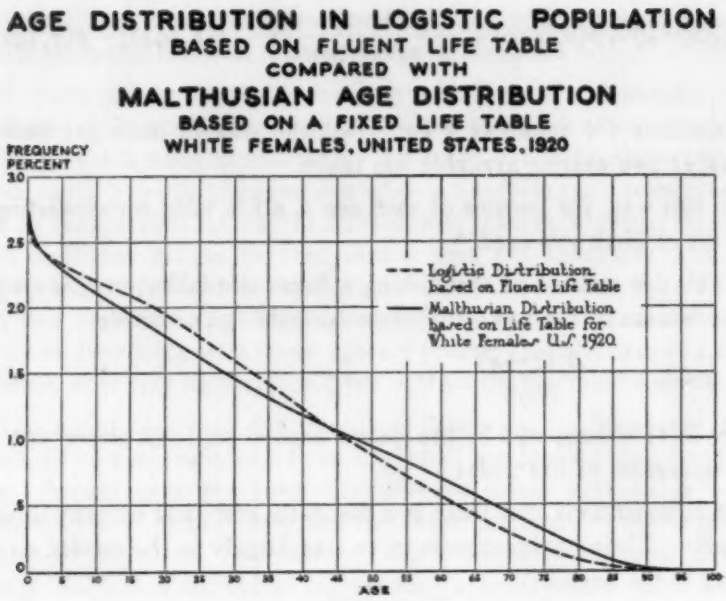
AGE DISTRIBUTION IN LOGISTIC POPULATION
BASED ON FLUENT LIFE TABLE
COMPARED WITH
MALTHUSIAN AGE DISTRIBUTION
BASED ON A FIXED LIFE TABLE
WHITE FEMALES, UNITED STATES, 1920
FREQUENCY PERCENT
3.0
2.5
2.0
1.5
1.0
.5
0
Logistic Distribution based on Fluent Life Table
Malthusian Distribution based on Life Table for White Females U.S. 1920
AGE

FIG. 10

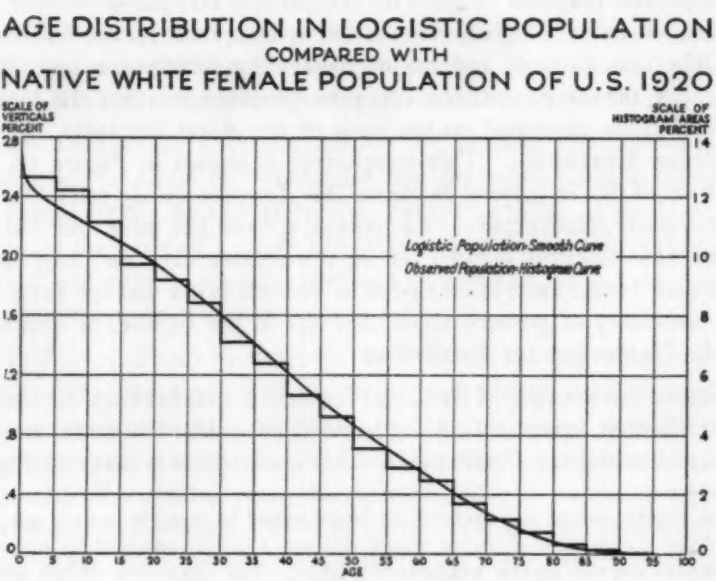
AGE DISTRIBUTION IN LOGISTIC POPULATION
COMPARED WITH
NATIVE WHITE FEMALE POPULATION OF U.S. 1920
SCALE OF VERTICALS PERCENT
SCALE OF HISTOGRAM AREAS PERCENT
2.8
2.4
2.0
1.6
1.2
.8
.4
0
1.4
1.2
1.0
.8
.6
.4
.2
0
Logistic Population Smooth Curve
Observed Population Histogram Curve
AGE

FIG. 11

11. It will be noted that the native [19] white female population of the United States conformed rather closely to the logistic age distribution thus computed.

Allowance for Immigration.—Lastly there remains to be considered the influence of immigration on some of the demographic characteristics as here computed.

TABLE 4

Comparison of Birthrate Computed According to Logistic Growth Curve and Observed Birthrate:

United States, White Females, Since 1915, per Thousand

(1)	(2)	(3)	(4)
YEAR	BIRTHRATE *		
	Computed		Observed
	Uncorrected for Immigration	Corrected for Immigration	
1915	28	25	25
1916	28	25	25
1917	27	24	24
1918	27	24	24
1919	26	23	22
1920	26	23	23
1921	26	23	24
1922	25	22	22
1923	25	22	22
1924	24	21	22
1925	24	21	21
1926	23	20	20
1927	23	20	20
1928	22	19	19
1929	22	19	19

* The birthrates here shown were actually computed on the basis of statistics for white females born per white female population. After the completion of the computation, decimals have been dropped, and to this degree of approximation it would be futile to retain a distinction between the rate "white female births per white female population" and the general birthrate in the form in which it is commonly stated.

[19] The "native" population was chosen for comparison, in order to eliminate at any rate in some degree the influence of immigration. A still further elimination of the effect of immigration is secured by making the comparison with the "native white of native parentage." It was found that this additional precaution had very little influence on the resulting picture.

"Birthrates" computed as described, from the growth curve of the population, include a contingent which is actually supplied not by births, but by net immigration. From the resulting series of birthrates it is therefore necessary to make a subtraction to correct for the fact that the actual growth included the effect of immigration. It was found that by making a constant subtraction of three per thousand per annum a set of figures was obtained which agreed very fairly with the observed birthrates, as shown in Table 4. This correction of three per thousand harmonizes well with the observed facts that during the period 1915 to 1928 the net female immigration was 2.4 per thousand per annum, and during the period 1908 to 1913 it was about five per thousand per annum. We may, if we please, regard this procedure merely as an empirical method of obtaining a series of birthrates, a method which is justified by its results over that period over which observed data for comparison are available.

COMPARISON OF POPULATION AS COMPUTED (A) BY THE LOGISTIC CURVE AND (B) BY SUMMATION OF THE SURVIVORS INTO EACH AGE OF LIFE

Method of Checking.—The convergence of the series developed in preceding pages and employed in the computations has not been subjected to a theoretical investigation, nor has a theoretical estimate been made of the magnitude of the remainder term when only a finite number of terms of the series are retained. Instead of such a theoretical investigation, an empirical arithmetical test has been made by comparing together the figures obtained for the *total population* (a) directly by the logistic formula and (b) by summation of the survivors into each year of life resulting from the annual births in accordance with the fundamental formula (1)

$$N(t) = \int_0^\infty B(t-a)\ p(a)da \qquad (1)$$

or its modification applicable to a fluent life table

$$N(t) = \int_0^\infty B(t-a)\ p(a,t-a)da \qquad (52)$$

The check in this form was applied separately to the case of a fixed life table, and also to the case of a fluent life table. The results obtained are shown below.

1. Check of Computation with Fixed Life Table

TABLE 5

Comparison of Population as Computed by
(a) Logistic Formula
(b) Summation of Age Distribution

YEAR	POPULATION COMPUTED BY		PER CENT ERROR
	(a) Logistic Curve	(b) Summation of Age Distribution	100 [(a)—(b)] / (a)
1850	23,245,464	23,255,366	— .04
1875	44,713,034	44,756,896	— .10
1900	77,220,510	77,254,191	— .04
1925	115,495,009	115,365,520	.11
1950	149,206,157	149,104,610	.07
1975	172,104,490	172,245,474	— .08
2000	185,049,703	185,136,134	— .05

The very good agreement shown in the figures of the table above represents, of course, merely an arithmetical check showing that the computation has been correctly conducted, and that the higher terms omitted in cutting off the infinite series at a finite term would make only a negligible contribution.

2. Check of Computation With Fluent Life Table

For the case of a fluent life table only one comparison of the total population computed by methods a). and b). was made, namely for the year 1920, as the computation becomes very laborious when a fluent life table is used.

The "fluent" life table was made by the method described in a previous section, interpolating the requisite values of $l_{x,t}$ between the tabular values as given in the following life tables.

a. England and Wales, Females No. 3—1838-54
b. England and Wales, Females No. 4—1871-80
c. England and Wales, Females No. 5—1881-90
d. United States White Females, Original Registration States, 1901
e. United States White Females, Original Registration States, 1910
f. United States White Females, Aggregate of 27 States and D. C. in 1919-20

Using the figures for the total births $B(t)$ obtained as described in a previous section by the use of successive life tables, and applying to these figures survival factors corresponding to the "fluent" life table as explained above, comparison was made between the total population computed by the summation formula (52), and by the logistic formula.

For the year 1920, this comparison gave the following result:

United States Population Computed by		*Discrepancy per cent*
Summation formula	Logistic formula	
105,212,000	107,875,000	2.5

There is here a discrepancy of 2½ per cent, which, considering the somewhat empirical nature of the method employed, and its admittedly approximate character, may be regarded as satisfactory.

APPENDIX

Alternative Expression for Annual Births.—The curve of annual births in a logistic population, under a fixed life table, is also capable of representation by an alternative formula which has the advantage of combining a certain simplicity with a high degree of approximation. It is a little more exact than the use of a *fundamental* component alone[20] of the exact formula given in an earlier section, and is simpler to use than the full series. It disregards, however, the slight asymmetry of the true curve.

The details of the development of this formula will here be omitted. The formula is

$$B(t) = \frac{GB_\infty}{1 + e^{-r(t-\eta-\theta)}} \tag{53}$$

$$B(t) = B_\infty \left\{ 1 - \frac{G}{1 + e^{r(t-\eta+\theta)}} \right\} \tag{54}$$

The formula (53) is to be used for values of t less than η, and the formula (54) for t greater than η, the origin of t being as before the

[20] The greatest discrepancy between the fundamental component alone and the full series is about 100,000. The greatest discrepancy between the alternative formula here given, and the full series is about 40,000.

center of the population growth curve, and the values of η, θ, G and β_1 being given by

$$\eta = \frac{1}{2r} log_e \frac{\beta_1}{b_1} \tag{55}$$

$$\theta = \frac{1}{r} [log_e\, b_0 - log_e(2\sqrt{b_1\beta_1} - b_0)] \tag{56}$$

$$G = \frac{\sqrt{b_1\beta_1}}{2\sqrt{b_1\beta_1} - b_0} \tag{57}$$

$$\beta_1 = \frac{1}{\int_0^\infty e^{ra} p(a)\, da} \tag{58}$$

Geometric Meaning of this Representation.—According to this mode of representation the curve of annual birth consists of two fragments of separate logistic curves pieced together. One of these logistics has its lower asymptote at zero, its upper asymptote at $G\,B_\infty$, and its center at $t = \eta + \theta$. The other has its lower asymptote at $(1 - G)B_\infty$, its upper asymptote at B_∞, and its center at $t = \eta - \theta$. The numerical values of the several constants, as computed for the United States, on the basis of the life table for 1919-20, are as follows:

$$\begin{aligned} G\,B_\infty &= 4.685 \text{ millions} \\ B_\infty &= 3.434 \text{ millions} \\ \eta &= -35.5 \text{ years} \quad \text{(zero at 1914.6)} \\ \theta &= 17.5 \text{ years} \\ b_0 &= .0174 \\ b_1 &= .0419 \\ \beta_1 &= .00449 \\ r &= .0314 \end{aligned} \tag{59}$$

Single Diagram to Represent Total Population, Annual Rate of Increase per Head, and Annual Births.—It can easily be shown that the rate of increase $r(t)$ per head of a population growing according to the logistic law follows an inverted logistic curve

$$r(t) = r_i \left(1 - \frac{N_t}{N_\infty}\right) \tag{60}$$

where r_i is the incipient rate of increase per head, which throughout the preceding sections has for brevity been written simply r without subscript.

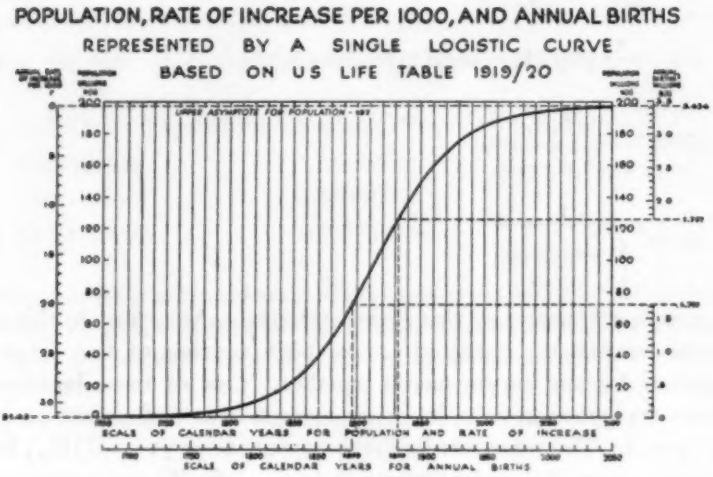
POPULATION, RATE OF INCREASE PER 1000, AND ANNUAL BIRTHS
REPRESENTED BY A SINGLE LOGISTIC CURVE
BASED ON U S LIFE TABLE 1919/20
SCALE OF CALENDAR YEARS FOR POPULATION AND RATE OF INCREASE
SCALE OF CALENDAR YEARS FOR ANNUAL BIRTHS

FIG. 12

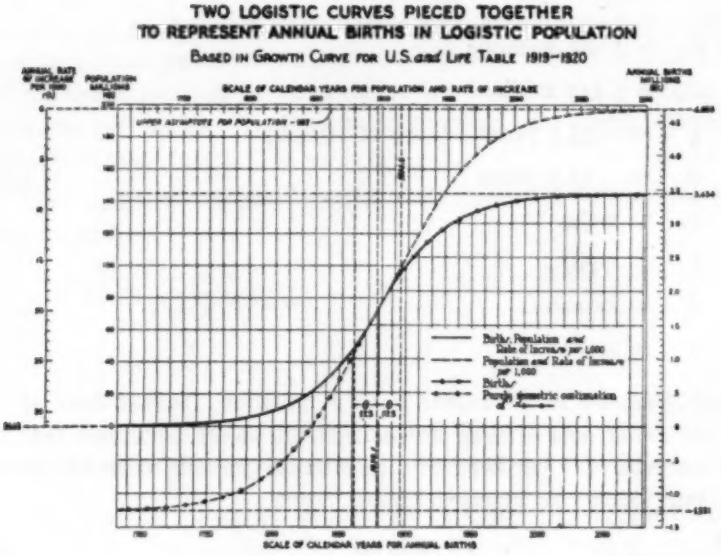
TWO LOGISTIC CURVES PIECED TOGETHER
TO REPRESENT ANNUAL BIRTHS IN LOGISTIC POPULATION
BASED IN GROWTH CURVE FOR U.S. 1919 LIFE TABLE 1919–1920
SCALE OF CALENDAR YEARS FOR POPULATION AND RATE OF INCREASE
SCALE OF CALENDAR YEARS FOR ANNUAL BIRTHS

FIG. 13

It has also been noted that the annual births $B(t)$ can be represented as two pieces of logistic joined together. This suggests that curves for all three of these demographic characteristics can be represented on a single diagram. This has been done in Figure 12. The basic feature of this diagram is the logistic curve for the population itself, $N(t)$. Values of this are read in the ordinary manner, the time scale running horizontally and the population scale vertically. To read values of the rate of increase per 1,000 a separate scale is shown at the extreme left and since this rate of increase follows an inverted logistic, this scale reads from above downwards.

To read values of the annual births $B(t)$ a split scale is used shown at the bottom of the diagram for the time scale and on the extreme right for the scale of births. Another mode of representation is shown in Figure 13. The diagrams practically explain themselves and further comment seems unnecessary.

SECULAR CHANGES IN MORTALITY RATES CONNECTED WITH CERTAIN ORGAN SYSTEMS*

BY ELIZABETH CARPENTER TANDY

Children's Bureau, United States Department of Labor

HISTORICAL BACKGROUND AND METHODS

Although there was probably never a time when man was not sufficiently interested in his own well-being to attempt to assign some cause for the death of his associates, the development of quantitative material adapted to the biological study of mortality has been a long slow process. There was no systematic collection of vital statistics whatsoever until the sixteenth century, and no attempt at an analysis of death records prior to the publication of "Natural and Political Observations Mentioned in the Following Index and made upon the Bills of Mortality," by Captain John Graunt, Citizen of London, in 1662. Since that first methodical examination of the bills of mortality, there has been a constant growth of interest in the registration of mortality and in the results of the careful scrutiny and analysis of vital statistics, which have come to form bodies of quantitative material available for study in medical institutions and public health departments.

Early mortality records were vague. The cause rubrics were mainly symptomological, and many terminal conditions could be equally well classified under any one of several heads. With the gradual development of medicine from an impressionistic art, in the sense that the diagnosis was little more than the impression of the practitioner, towards a science based upon actual knowledge of normal and pathological tissues, upon laboratory findings, and upon the careful record of physiological signs, came the differentiation and naming of diseases which had

* From the Institute for Biological Research and the Departments of Biology and Biostatistics of the School of Hygiene and Public Health, Johns Hopkins University. This study was accepted in partial fulfilment of the requirements for the degree of Doctor of Science in Hygiene. In preparing the material for publication it has been necessary, because of the exigencies of space and expense, to make considerable deletions from the original dissertation, which is filed in the archives of the School of Hygiene. The work was completed in May, 1926.

previously been unrecognized as lethal entities. The many forward steps of medicine thus led to and made possible the demand for concise, mutually exclusive, and uniform cause titles in the mortality bills.

By the time of William Farr (1807-1883) there was a fair amount of appreciation of the need for improvement in mortality registration in England. We find him emphatically stating in his First Annual Report to the Registrar General that "nomenclature is of as much importance to this department of inquiry, as weights and measures in the physical sciences, and should be settled without delay." Constant study and consideration of the problems of nomenclature and classification during the next few years brought system and order out of chaos in that country. About the same time much attention was directed toward nosology in many parts of continental Europe. By 1855 the International Classification of the Causes of Death had been developed. Provision was made for its decennial revisions at a somewhat later date. This classification formed a foundation for uniform mortality statistics and made possible the comparison of the vital facts of different localities.

The International Classification, for statistical purposes, rather rigidly defines and separates the causes of human mortality into something over 180 distinct units. The individual rubrics, at the time of writing this paper, are grouped into fourteen general classes. These are:

I. General diseases.
II. Diseases of the nervous system and organs of special sense.
III. Diseases of the circulatory system.
IV. Diseases of the respiratory system.
V. Diseases of the digestive system.
VI. Non-venereal diseases of the genito-urinary system and annexa.
VII. The puerperal state.
VIII. Diseases of the skin and the cellular tissue.
IX. Diseases of the bones and the organs of locomotion.
X. Malformation.
XI. Early infancy.
XII. Old age.
XIII. External causes.
XIV. Ill-defined diseases.

The most superficial examination of these summary categories indicates that there was no attempt to formulate a system based upon a

single underlying idea. The concept of the planners is probably well expressed by Farr in his Sixteenth Annual Report, page 76. It is:

> Classification is a method of generalization. Several classifications may, therefore, be used with advantage; and the physician, the pathologist, or the jurist, each from his own point of view, may legitimately classify the diseases and the causes of death in the way he thinks best adapted to facilitate his inquiries, and to yield general results.
>
> The medical practitioner may found his main divisions of diseases on their treatment as medical and surgical; the pathologist on the nature of the morbid action or product; the anatomist, or the physiologist on the tissues or organs involved; the medical jurist, on the suddenness or the slowness of death; and all those points well deserve attention in a statistical classification.

It is patent from this statement that rearrangement of the data of vital statistics has been recognized from the beginning as necessary for special studies.

The present study of mortality records is based upon Pearl's biological classification of the causes of death (3, pp. 140-176). It is pertinent, therefore, to describe his scheme of rearrangement of the rubrics of the international code and to summarize the underlying biological principles which form its scientific ground work.

Pearl's classification is founded upon the following general biological considerations:

1. Natural death as distinguished from accidental death is preceded by definite structural and functional changes.

2. In the evolution of higher organisms there has been a progressive setting apart of cells and tissues to perform particular vital functions with a consequent loss of ability to perform all vital functions independently.

3. Because of this differentiation and specialization the parts of the multicellular organism are mutually dependent upon each other to keep themselves going.

4. As soon as any one of the cell groups or tissues of the multicellular organisms begins, for any reason whatsoever, to fail to perform its special function properly, it upsets the delicate balance of the whole associated community of cells and tissues.

5. Any disturbance in the balance which is not properly righted by some regulatory process eventually results in the death of the organism.

Specifically, the fundamental idea of the biological classification is the grouping of as many as possible of the causes of death under the several organ systems of the body, the functional breakdown of which,

from whatever immediate cause, results in the cessation of life. Its logical foundation is the fact that, biologically considered, death results because some organ system or group of organ systems primarily fails to continue its normal functions. A large majority of the statistically recognized causes of death of the International Classification can be assigned places under organological rubrics. The small residue of causes which cannot be so assigned, for one reason or another, are placed together in a class designated as "All other causes of death."

The headings of Pearl's biological classification are:

I. Circulatory system, blood and blood-forming organs.
II. Respiratory system.
III. Primary and secondary sex organs.
IV. Kidneys and related excretory organs.
V. Skeletal and muscular systems.
VI. Alimentary tract and associated organs concerned in metabolism.
VII. Nervous system and sense organs.
VIII. Skin.
IX. Endocrine system.
X. All other causes of death.

The justification for the inclusion of the several cause titles under the organological heads is fully discussed in the chapter on "Evolution and Mortality" in Pearl's "Studies in Human Biology" (3, pp. 146-161). It will not be repeated here.

The primary object of the present work is to investigate the *time changes in mortality* assignable to five of the organological rubrics listed above. The rubrics of the International code are grouped under the organ systems exactly as in Pearl's original account cited, with the exception of diabetes, which has been transferred from Group VI (Alimentary tract and organs associated with metabolism) to Group IX (Endocrine system) for obvious reasons. The five rubrics omitted from this present discussion are Group III (primary and secondary sex organs); Group V (Skeletal and muscular system); Group VIII (Skin); Group IX (Endocrine system); and Group X (All other causes). In actual fact these were tabulated, but for a number of reasons it appears desirable in the published report to confine attention to the other five rubrics, which caused, in the years and demographic groups here studied, some 88.5 per cent of the total mortality capable of biological classification, on the average.

Two sets of comparisons are made in the study: one between the urban and rural portions of the U. S. Registration Area; the other between the white population of the Registration States of 1911—constituted as defined below—and the population of England and Wales. In both cases the comparisons extend over a period of years. These demographic groups with one exception—that of the U. S. Registration States of 1911 (white population)—are frequently used in biostatistical study, and hence need no explanation. The U. S. Registration States of 1911 (white population) group consists of the *total* population of the States in the Registration Area that year which had less than 10 per cent negro inhabitants at the time of the 1910 Census, plus the *white* population only of the States in the Area showing more than 10 per cent negro population at the time of that Census. The States of the U. S. Registration Area in 1911 are: California, Colorado, Connecticut, Indiana, Kentucky, Maine, Maryland, Massachusetts, Michigan, Minnesota, Montana, New Hampshire, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Utah, Vermont, Washington and Wisconsin. Of these states Kentucky and Maryland showed more than 10 per cent negro population at the 1910 Census.

The material consists of the crude annual death rates by causes of the urban and rural portions of the U. S. Registration Area, for the years 1910-1920 (inclusive); and of England and Wales for the years 1911-1922 (inclusive). The rates are in terms of deaths per 100,000 population.

The sources of the material are the United States Census annual volumes of Mortality Statistics, and the Annual Reports of the Registrar General of England and Wales, for the years included in the study. The data appear in those volumes in the form of crude annual death rates, except in the case of the U. S. Registration States of 1911 (white population). For that group the actual deaths recorded under each rubric of the International Classification were extracted separately for each State in the list. Only white deaths were taken in the States showing more than 10 per cent negro population in the 1910 Census. The actual deaths were totaled by years for each rubric and the crude annual death rates of the several cause titles calculated according to the formula:

$$\text{Crude death rate} = \frac{\text{Actual deaths} \times 100{,}000}{\text{Total population}}$$

The crude death rates of the cause titles appearing in the reports and those obtained by the formula were tabulated by years for each rubric in the organological classification. The rates of the individual rubrics were then summarized by years for each organ system class. Crude annual death rates specific for each organ system were thus gained for each demographic group included in the study.

The first step in the analysis of the material was the plotting of the annual rates of the several organ system classes on semi-logarithmic paper. The rates for the mortality assignable to the same organ system in the two comparable demographic groups are plotted on the same diagram in all cases. To bring out the general tendency toward a constant rate of change a straight line of best fit is calculated by least squares and superimposed upon the observed figures. These trend lines are obtained by the equation:

$$\log y = a + bx \qquad (1)$$

in which y is the death rate and x is the time.

The equation is equivalent to:

$$y = 10^{a+bx} = 10^{a} \cdot (10^{b})^{x}$$

or $$y = A(B)^{x} \qquad (2)$$

where $(A = 10^{a})$ and $(B = 10^{b})$

or $(\log A = a)$ and $(\log B = b)$ (3)

This is the ordinary equation for geometric progression. It states that the death rate for any year is equal to the death rate of the preceding year multiplied by B. Obviously, if B is greater than 1 the death rate is increasing with time, and if B is less than 1, the death rate is decreasing. A is the death rate of the year when $x = 0$. In this study the origin of x is taken as the year immediately preceding the first year of the observations.

The values of the parameters are obtained by solving the normal equations:

$$na + b\Sigma x = \Sigma (\log y)$$
$$a\Sigma x + b\Sigma x^{2} = \Sigma (x \log y) \qquad (4)$$

$\Sigma (\log y)$ is the summation of the logarithms of the observed death rates. $\Sigma (x \log y)$ is the summation of the logarithms of the observed death rates multiplied respectively by the x value of the year of occurrence. n is the number of years included in the study. The values of a and b that come from the equations (4) give us the values of A and B through the use of the relationship between these constants previously stated in equations (3).

A figure permitting judgment as to the significance of the value of the B constant for each curve is obtained in terms of Yule's formula for the standard error of a coefficient of regression. The formula as used is:

$$\text{Probable error of } b = .67449 \frac{\sigma_{y \cdot x}}{\sigma_{x} \sqrt{n-2}}$$

Because of the small number of years involved in the data $\sqrt{n-2}$ is used in the above formula in place of $\sqrt{n}$.

The probable error of B was obtained from the probable error of b through the relationship of the two constants: i. e. ($\log B = b$).

$$\text{P. E. of } B = \frac{\text{antilog of } (b + \text{P.E.}_b) - \text{antilog of } (b - \text{P.E.}_b)}{2}$$

This equation neglects the skewness due to the logarithmic scale but since the variation of b is small with respect to b this skewness factor is insignificant. The B values and their probable errors were expressed in percentages.

Mere comparison of the several curves, one with another, which have trend lines superimposed upon the observed data, indicates that some degree of relationship obtains between the synchronous deviations from the trends plotted on the logarithmic scale. This seems to be of two types: (a) association between the synchronous deviations occurring in the mortality assignable to the same organ system in the compared populations; (b) association between the synchronous deviations occurring in a single population in the mortality assignable to different organ systems. The first type suggests that unusual temporary factors operating at the same time in the compared populations affect the death rate assignable to particular organ systems in both populations in a congruous manner; the second, that the mutually dependent organ systems may be more or less simultaneously affected by unusual conditions and that the result of the temporary change in the environment appears in the biologically classified death rates.

The usual Pearsonian method of correlation was used to study the degree of association between the synchronous deviations. The rate of change concept was preserved by using the logarithms of the deviations. The degree of association was analyzed between the synchronous deviations in (1) the same organ system in the comparable areas, (2) the more important different organ systems in the same area. The formulae for the coefficient of correlation (r) and the probable error of the coefficient are:

$$\text{Coefficient of correlation } r = \frac{\Sigma (xy)}{n \cdot \sigma_x \cdot \sigma_y}$$

$$\text{Probable error of } r = .67449 \frac{1 - r^2}{\sqrt{n - 2}}$$

The next point that demands consideration is that of obtaining a series of generalized figures which will indicate the importance of the death rates assignable to each organ system in one population in relation to those assignable to the same organ system in the comparable population. Such constants were obtained in terms of the ratio of the death rates assignable to an organ system in one population to the death rates assignable to the same organ system in the other population.

If Y_1 represents one death rate and Y_2 the other to be compared, and their time trend equations are:

$$\log Y_1 = a_1 + b_1 (x)$$
$$\text{and } \log Y_2 = a_2 + b_2 (x)$$

Then

$$\text{log of the ratio} = \log \frac{Y_1}{Y_2} = \log Y_1 - \log Y_2 = (a_1 - a_2) + (b_1 - b_2)\, x$$

The values substituted are those previously obtained by fitting the straight trend lines.

The observed ratios for each year of the experience are next secured for the actual data.

$$\text{Ratio} = \frac{\text{Observed death rate of the year (series 1)}}{\text{Observed death rate of the year (series 2)}}$$

The logarithm of the probable error of the ratio is then computed in the ordinary way by using the sums of the squares of the deviations of the calculated and observed ratios expressed in logarithmic form.

The probable error of the rate of change of the ratio is then determined from the probable errors of b_1 and b_2 by the usual method of determining the probable error of the difference of two constants. The rate of change constants and their probable errors are expressed in percentage form.

It seems desirable to select a central value of each curve which will serve both as an index of the ratio curves, and as an index of the mortality curves. For this purpose the geometric mean has been chosen. Now the smoothed mid-year figure, that of 1915, in each of the several series for the Urban and Rural portions of the U. S. Registration Area is the geometric mean of that material, but no such value is available for the England and Wales and the U. S. Registration States of 1911 (white population), since the period included in the study of these areas involves an even number of years. However, the year 1915 only slightly precedes the mid-point of the series, and since the geometric mean minimizes the effect of the variations of the marginal observations, the smoothed figures of that year are deemed sufficiently satisfactory indices. The 1915 values of the England and Wales and the U. S. Registration States of 1911 (white population) smoothed curves are in fact the weighted geometric means of the material. The calculated values of 1915 are therefore used as indices in comparing the several ratio relationships, and in showing the relative importance of the different organ systems in mortality.

THE RELATIVE IMPORTANCE OF THE DIFFERENT ORGAN SYSTEMS IN MORTALITY

When death is considered from a biological point of view mortality rates take on added interest. For the causes of death are no longer

expressed in heterogeneous and therefore meaningless groups, but summarized in broad basic categories which have biological pertinence. Galton once planned a study of the duration of human life and stated his fundamental conception in these terms: (4, p. 350.)

> The strength and enduring quality of the body, like that of a chain, must be measured at its weakest point; and though in it more than in a chain, the strength and the quality of some parts may compensate for deficiency in others, yet the very opposite may be the result. The stronger organs may relieve, but they may also oppress the weaker members. A strong digestive system may overload a weakly circulation, and prove injurious to the liver, lungs or kidneys, in fact a disturbing agent to the general nutrition. The requisite for longevity, therefore, we may not expect to be so much strength of organs as their enduring quality, a good mutual adjustment, in other words a good balance.

Table 1 shows the general results obtained regarding the mortality in the five organological classes and the different populations included in this study. As previously stated these figures are the calculated death rates from the fitted trend lines for the year 1915. They are generalized centering constants for the respective time period covered. Fig. 1 visualizes the relative importance of the mortality associated with five different organ systems in the several populations. The rates of Table 1 have been arranged in descending order of magnitude.

Two-thirds of the total mortality is accounted for by causes primarily connected with the respiratory and circulatory systems and the

TABLE 1

Showing the Relative Importance of Different Organ Systems in Mortality Death Rates of 1915 from Fitted Trend Lines

CLASS NO.	ORGAN SYSTEM	DEATH RATES PER 100,000			
		Registration Area, U. S. A.		U. S. Registration States of 1911 (whites)	England and Wales
		Urban	Rural		
II	Respiratory system	381.4	303.5	324.3	403.9
I	Circulatory system, blood	326.4	292.6	315.7	304.5
VI	Alimentary tract and associated organs	266.1	219.9	236.9	230.5
IV	Kidneys and related excretory organs	119.4	86.3	102.8	45.2
VII	Nervous system and sense organs	85.7	79.2	82.5	103.3

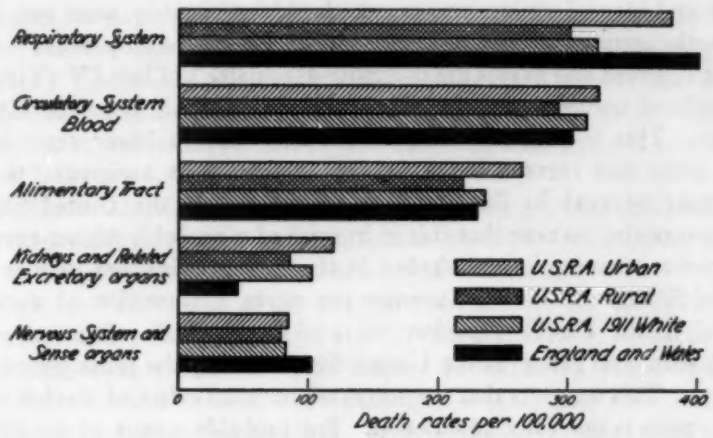

FIG. 1. RELATIVE IMPORTANCE OF FIVE ORGAN SYSTEMS IN MORTALITY

alimentary tract. At the top of the list stands the respiratory system. On first thought this might seem to be merely a result of the influenza epidemic of 1918. However that epidemic year was eliminated from consideration in the analysis of the data and the calculated rate for 1915, which would have been greatly augmented by the inclusion of the respiratory deaths of 1918, was actually reduced somewhat below normal by the low rates which followed the pandemic.*

Next to the mortality assignable to the respiratory system stands that associated with the circulatory system. Third comes the mortality associated with the alimentary tract. When Pearl analyzed the death rates of the U. S. Registration Area 1901-1910, England and Wales 1914, and Sao Paulo 1917, he found that the respiratory system and the alimentary tract together accounted for more than half of the deaths biologically classifiable in all the populations studied; and that the mortality connected with the alimentary tract and associated organs was relatively more important than that connected with the circulatory system. He tentatively concluded that this was to be expected since the respiratory system and the alimentary tract were exposed directly to environmental influences in the performance of their functions. The past few years, however, have seen much improvement in child care and general sanitary measures. The result has been a great reduction in the specific death rates during the younger age periods. The circulatory system,

* For a discussion of the reasons for this elimination, see pages 508-509.

blood and blood-forming organs, which must ultimately wear out, consequently assume a more important position in the relative scale.

In England and Wales the mortality assignable to Class IV (Kidneys and related excretory organs) is much lower than it is in the United States. This is primarily and immediately due to lower rates from both acute and chronic nephritis (the major items assignable to the excretory system) in England and Wales than in the United States. It is interesting to note that this is in spite of a probably higher average per capita consumption of alcohol in the United Kingdom than in the United States. At least the average per capita consumption of absolute alcohol in the United Kingdom 1894-1898 was 2.08 gallons as compared with 1.00 gallon in the United States during the same period (5, p. 613). This suggests that the supposed harmful effect of alcohol upon the kidneys is not fully understood. But probably a part of the difference between the American and British death rates under Class IV may be due to differences in registration, and perhaps diagnostic, customs in the two countries. It seems incredible that there can actually exist so great a difference in the incidence of nephritis, acute and chronic, in the two countries.

Causes of death connected with the nervous system are responsible for 6 or 7 per cent of all the deaths in all the populations. The English rate is higher than that of the United States in all the cause titles of this class with the noteworthy exceptions of deaths from suicide and from alcoholism, in which the American rates exceed the British.

SECULAR CHANGES IN DEATH RATES IN THE URBAN AND RURAL U. S. REGISTRATION AREAS, 1910-1920

The difference between urban and rural mortality has been discussed since the earliest days of vital statistics. Graunt noted the preponderance of urban over rural mortality. Farr gave much attention to the subject. Those who first established the high mortality of urban districts considered cities "the graves of mankind." "The reason for the baneful influence of great towns as it has now been exhibited" (9, p. xiii), Doctor Price, the founder of the Equitable Insurance Company, emphatically stated, "are plainly—first, the irregular modes of life, the luxuries, debaucheries, and pernicious customs, which prevail more in towns than in the country. Secondly, the foulness of the air in towns occasioned by uncleanliness, smoke, the perspiration and breath of inhabitants, and the putrid streams from drains, churchyards, kennels

and common sewers." Farr himself thought that there was reason to believe that the aggregation of mankind in towns was not inevitably disastrous. "The causes of mortality in towns are well known," he wrote, in his Third Annual Report (1, p. 170), and went on to say that "it cannot be too frequently repeated that they admit of removal to a great extent."

Public health administration and the gradually growing adaptation of man to city life have done much to reduce the difference between urban and rural mortality, but urban rates are still in excess.

The material now to be discussed is furnished by the death rates of the urban and rural portions of the U. S. Registration Area, for the years 1910-1920 inclusive. The Census definition of urban districts includes towns and cities of more than 2500 inhabitants. The less populous areas are classified as rural. It is obvious that the urban group is not a homogeneous one, and this is particularly true so far as concerns public health and sanitary measures, and the sanitation of large cities is often superior to that of the medium sized towns. In urban districts of less than 5000 population, satisfactory water supply, proper waste disposal, and adequate local medical facilities are sometimes lacking.

The three methods of approach to the question of the biological aspects of time changes in death rates in the urban and rural districts of the United States during the time period 1910-1920, which have been described above, are:

(1) The ratio of urban and rural death rates.
(2) The trend of the rates and the annual percentage rate of increase or decrease in value.
(3) The correlation of the synchronous deviations from the trend in the two populations.

The data are presented in Tables 2-6 inclusive.

Considering now the mortality assignable to the first biological class, namely the death rates due to the breakdown of the *Circulatory system, blood and associated organs,* we note that the urban rates are above the rural. But the difference is not great. The ratio of the two, Urban/Rural, is $1.162 \pm .027$. The curves have a considerable degree of similarity. There was but little secular change in the rates during the time period covered. In the urban population the annual percentage rate of change in the death rates is $+ .391 \pm .256$. That for the rural population is $- .426 \pm .299$. Neither of these figures is significant,

TABLE 2

Showing the Crude Annual Death Rates per 100,000 Living for Five Organological Cause Groups in the Urban Portions of the U. S. Registration Area, 1910-1920 inclusive

CLASS NO.	ORGAN SYSTEM	YEARS 1910	1911	1912	1913	1914	1915	1916	1917	1918	1919	1920
I	Circulatory system, blood	320.2	318.6	320.8	324.2	320.4	317.4	340.8	346.5	347.1	306.4	330.0
II	Respiratory system	418.2	384.8	365.8	370.6	359.8	362.0	376.4	390.8	875.5	399.4	389.6
IV	Kidneys and related excretory organs	117.1	118.2	122.5	124.1	123.3	122.3	127.4	128.8	119.3	107.2	105.2
VI	Alimentary tract and associated organs	329.8	298.3	285.9	292.4	272.4	255.8	261.0	256.9	251.5	219.9	222.9
VII	Nervous system and sense organs	103.6	98.0	93.4	95.0	89.2	83.6	96.2	84.4	77.5	66.4	64.9

TABLE 3

Showing the Crude Annual Death Rates per 100,000 Living for Five Organological Cause Groups in the Rural Portions of the U. S. Registration Area, 1910-1920 inclusive

CLASS NO.	ORGAN SYSTEM	YEARS 1910	1911	1912	1913	1914	1915	1916	1917	1918	1919	1920
I	Circulatory system, blood	302.2	290.6	289.3	290.8	292.0	299.0	304.4	314.2	291.9	261.3	286.5
II	Respiratory system	300.8	303.2	283.5	282.9	274.3	286.7	304.7	308.7	684.9	354.2	330.1
IV	Kidneys and related excretory organs	83.2	80.8	87.3	86.8	87.8	92.8	92.2	94.2	85.1	78.8	81.9
VI	Alimentary tract and associated organs	272.8	241.6	227.6	244.6	225.0	210.2	223.4	224.2	211.9	180.1	175.1
VII	Nervous system and sense organs	101.8	89.6	87.0	86.8	83.0	80.3	79.6	74.9	75.6	62.8	58.9

TABLE 4

Showing the Ratios of the Death Rates for Five Organological Cause Groups of Causes in the Urban and Rural Portions of the U. S. Registration Areas, 1915 Values

CLASS NO.	ORGAN SYSTEM	RATIO $\frac{\text{URBAN}}{\text{RURAL}}$	ANNUAL PERCENTAGE RATE OF CHANGE OF RATE
I	Circulatory system, blood	1.162 ± .027	+ .820 ± .397
II	Respiratory system	1.167 ± .025	— 1.474 ± .528
IV	Kidneys and related excretory organs	1.330 ± .024	— .778 ± .558
VI	Alimentary tract and associated organs	1.206 ± .026	— .072 ± .458
VII	Nervous system and sense organs	1.097 ± .037	+ .282 ± .574

TABLE 5

Showing the Annual Percentage Rate of Change in the Mortality Rate for Five Organological Cause Groups in the Urban and Rural Portions of the U. S. Registration Areas 1910-1920 Inclusive

CLASS NO.	ORGAN SYSTEM	URBAN	RURAL	DIFFERENCE URBAN—RURAL
I	Circulatory system, blood	+ .391 ± .256	— .426 ± .299	+ .817 ± .393
II	Respiratory system	+ .071 ± .342	+ 1.567 ± .415	— 1.497 ± .538
IV	Kidneys and related excretory organs	— .815 ± .395	— .037 ± .398	— .778 ± .560
VI	Alimentary tract and associated organs	— 3.453 ± .227	— 3.384 ± .381	— .069 ± .443
VII	Nervous system and sense organs	— 4.105 ± .442	— 4.375 ± .325	+ .270 ± .548

TABLE 6

Showing the Coefficients of Correlation of the Logarithms of the Synchronous Deviations of the Observed and Calculated Death Rates Assignable to the Same Organ Systems in Different Populations, for Five Organological Cause Groups, in the Urban and Rural Portions of the U. S. Registration Areas 1910-1920 Inclusive

CLASS NO.	ORGAN SYSTEM	COEFFICIENT OF CORRELATION
I	Circulatory system, blood	+ .828 ± .071
II	Respiratory system	+ .849 ± .066
IV	Kidneys and related excretory organs	+ .903 ± .042
VI	Alimentary tract and associated organs	+ .909 ± .039
VII	Nervous system and sense organs	+ .716 ± .110

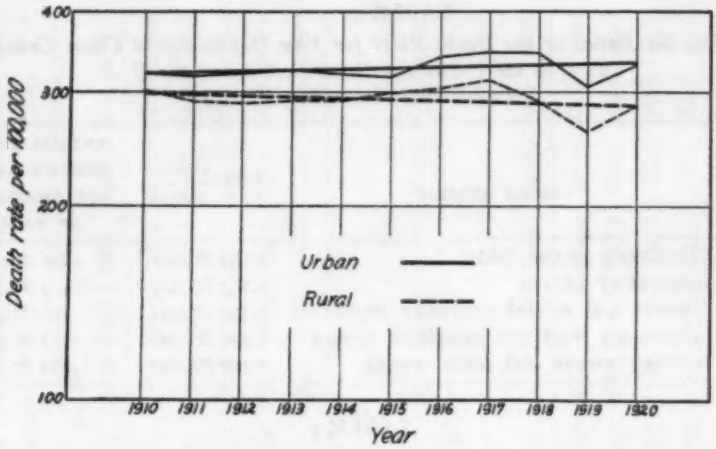

FIG. 2. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS I (CIRCULATORY SYSTEM, BLOOD) IN THE URBAN AND RURAL PORTIONS OF THE U. S. REGISTRATION AREA 1910-1920 INCLUSIVE

in comparison with the probable errors. There is indication of a slight tendency toward increase in the urban population and decrease in the rural, but neither trend can be considered to be significant. The deviations from the trend lines in the two curves are remarkably similar. In general, both sets of rates are below the trend line during the first few years of the period. From 1915 onward to the year following the influenza epidemic they stand slightly in excess of the norm. In 1919 there is a drop and in 1920 a return of the death rates to the usual position. The synchronous deviations are identical in direction and proportionally similar in the two populations in the several years examined. Correlating the synchronous deviations from the trend we have $r = + .828 \pm .071$. The factors which have caused variation in the urban and rural circulatory system death rates at each point of time have resulted in similar and proportional increases and decreases.

The *Respiratory system*, which was responsible for more deaths than any other in all four populations studied, presents serious difficulties in the particular period studied. These difficulties lie in the experience of the later years of the period. Prior to 1915 there apparently was in both urban and rural districts a tendency towards a decrease in mortality from respiratory diseases. But following that year a steady tendency to rise anticipated the influenza epidemic of 1918, when death rates

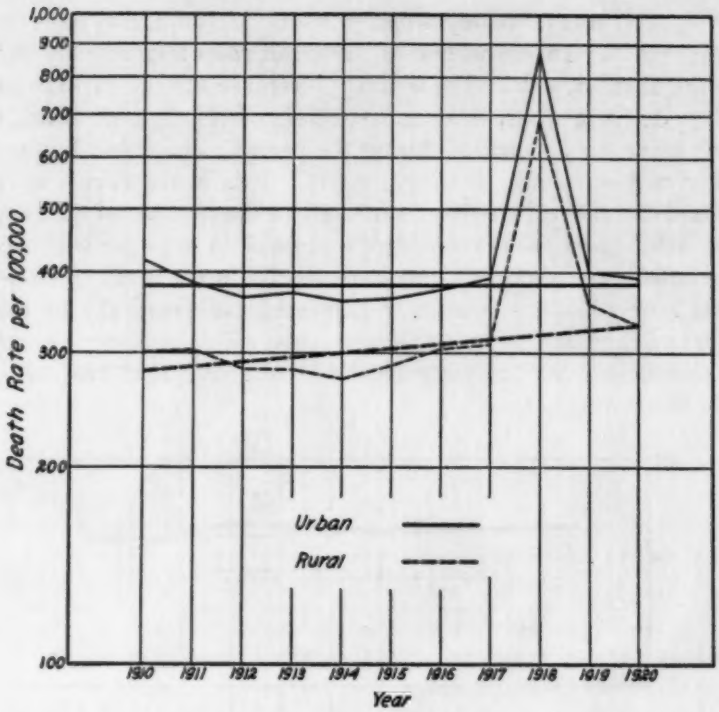

FIG. 3. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS II (RESPIRATORY SYSTEM) IN THE URBAN AND RURAL PORTIONS OF THE U. S. REGISTRATION AREA 1910-1920 INCLUSIVE

assignable to the respiratory system reached a maximum point unprecedented in the history of vital records. The next year there was a decline, but both 1919 and 1920 show rates in excess of those of the years immediately preceding the pandemic. The rates were so upset during the epidemic that years must pass before an entirely adequate analysis of the trends of respiratory death rates can be made. It would, however, be an inadequate study of the biological aspects of mortality rates should we entirely disregard the most important systemic group of the classification. The alternative, that of eliminating the year 1918 from consideration, which permits of a trend line that expresses the general characteristics of the rates of the other years of the period, seems the only possible method of approach. This elimination is therefore made and the rates of the non-epidemic years are analyzed.

The ratio in the non-epidemic years of Urban/Rural mortality is 1.167 ± .025. The trend line of the urban rates is practically parallel with the abscissal axis. The annual percentage change in the rates is + .071 ± .342 which is an entirely insignificant figure. Rural rates on the other hand increased during the period as indicated by the percentage rate of change + 1.567 ± .415. It is in the deviations from the trend that the really striking similarity of the two sets of data appear. When urban rates fall below their norm, rural do likewise; with the rise in the urban rates there is a simultaneous rise in the rural. The degree of this synchronous association is expressed mathematically by the coefficient of correlation $r = + .849 \pm .066$. This indicates a considerable association; but for the exclusion of the year 1918 the coefficient would be even higher.

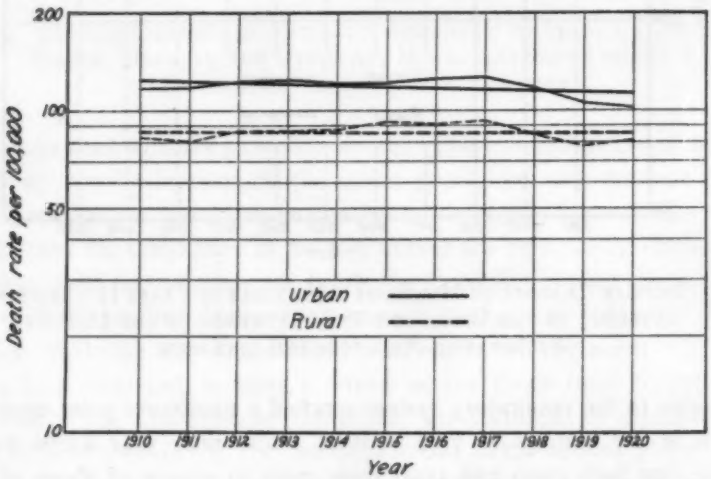

FIG. 4. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS IV (KIDNEYS AND RELATED EXCRETORY ORGANS) IN THE URBAN AND RURAL PORTIONS OF THE U. S. REGISTRATION AREA 1910-1920 INCLUSIVE

Class IV (*Kidneys and related excretory organs*) is made up of but few cause titles. That it is highly important in the death toll has been demonstrated in an earlier part of this paper.

In these curves of Fig. 4 we again see urban mortality rates in excess of rural. The ratio, Urban/Rural, is 1.330 ± .024. To the eye the trend lines of both curves are practically parallel to the base. The annual percentage rate of change in the urban population is —.815 ± .395;

in the rural $-.037 \pm .398$. Both figures are low and insignificant. Mortality assignable to the excretory organs may be said to have been remarkably stable during the period covered in this study. There were only small deviations from the trend in both populations. Those occurring at the same point of time were identical in direction and proportional in magnitude. The coefficient expressing the relationship is $r = + .903 \pm .042$.

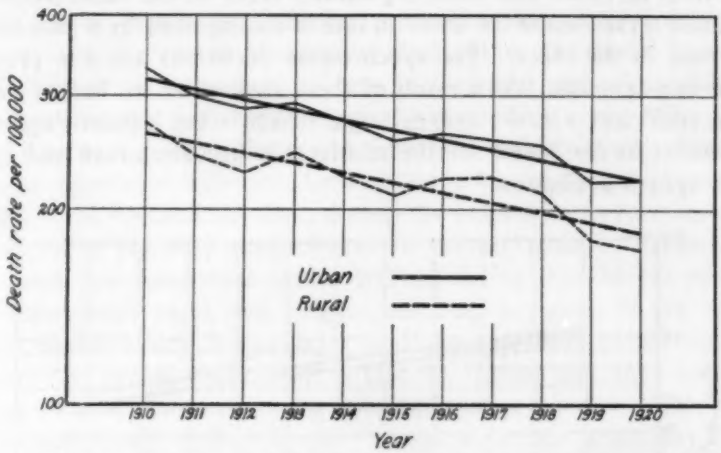

FIG. 5. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS VI (ALIMENTARY TRACT AND ASSOCIATED ORGANS) IN THE URBAN AND RURAL PORTIONS OF THE U. S. REGISTRATION AREA 1910-1920 INCLUSIVE

Class VI (*Alimentary tract and associated organs concerned in metabolism*) is interesting not only because the rate of mortality assignable to this organ system forms a considerable portion of the total mortality, but also because deaths from every age period of life are represented in this class, and because it is toward the prevention of this mortality that a considerable part of all public health effort is primarily directed. In this rubric is included typhoid fever, which has had an enormous decrease since efforts have been directed toward purification of water and milk supplies and proper sewage disposal. Also diarrhea and enteritis are in Class VI, where the same factors have helped to bring about a great reduction in mortality.

As in all the previously discussed rubrics we see urban rates in excess of rural. The ratio, Urban/Rural, is $1.206 \pm .026$. The trend curves of the rates show a marked decline in both urban and rural areas. The

urban percentage rate of change is $-3.453 \pm .227$ and the rural $-3.384 \pm .381$. There is no significant difference between the two rates of decrease. This seems rather remarkable when we consider the relative amount and quality of public health efforts, in the period here under review, in the urban as compared with the rural portions of the U. S. Registration Area.

The deviations from the trend lines alternate above and below. The high rates in urban and rural populations occur at the same points of time, and a rate below the trend in one is accompanied by a rate below the trend in the other. The synchronous deviations are also proportional in magnitude. As a result of these similarities we have a correlation coefficient $r = + .909 \pm .039$, which is the highest degree of association so far found for the relationship between urban and rural organ system deviations.

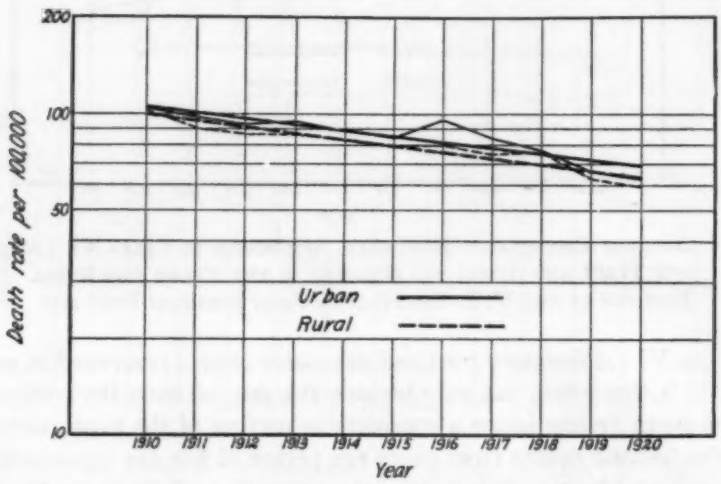

FIG. 6. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS VII (NERVOUS SYSTEM AND SENSE ORGANS) IN THE URBAN AND RURAL PORTION OF THE U. S. REGISTRATION AREA 1910-1920 INCLUSIVE

The mortality assignable to the *Nervous system and sense organs* results from a considerable series of single causes, each of which has a relatively low death rate, but which as a whole are responsible for a fair proportion of the total mortality. With the exception of meningitis the mortality assignable to this Class VII falls mainly in the older age periods of life.

While the urban rate is again higher than the rural, the difference between them is small, the ratio, Urban/Rural, being $1.097 \pm .037$, which does not differ significantly from unity. The curves are very close together on the graph. Both curves have a definite downward trend. The annual percentage rate of decrease for the urban population is $-4.105 \pm .442$. For the rural population the percentage rate of decrease is $-4.375 \pm .325$. The mortality assignable to the nervous system and sense organs thus exhibited, during the years covered in this study (1910-1920 inclusive), the most rapid rate of decline of any of the five organ systems here studied. Why this should have been so is difficult to understand. There is a general and widespread impression that the rush and hurly-burly of modern urban life is hard on the nervous system. But the data seem to indicate clearly that there was no significant difference between the urban and rural populations of the U. S. Registration Area, during the years 1910 to 1920 inclusive, in respect of mortality assignable to the nervous system. Furthermore the death rate from these causes declined during these eleven years at the same rather rapid rate. Again, one does not know of any special public health efforts having been made against these causes of death during this period. It is possible that the decline may have been due to uniform and universal changes in registration practice. This possibility must always be kept in mind, of course, but the probability that it was an important factor in the case seems small. It would seem to require a degree and extent of concerted action amongst physicians and registrars which, so far as one knows, did not in fact exist.

SUMMARY REGARDING URBAN AND RURAL MORTALITY

The more significant observations in the comparison of the time changes in mortality assignable to each of five organ systems in the urban and rural populations of the U. S. Registration Area, 1910-1920, may be summarized as follows:

(1) Urban death rates are in excess of rural in every organ system class studied. There has been no evidence of a significant change in the ratio of Urban/Rural death rates in any of the five more important organ system rubrics.

(2) Changes with time in the death rates assignable to the several organ systems in urban and rural populations during the period studied were of the following character:

	Urban	*Rural*
I. Circulatory	No significant change	No significant change
II. Respiratory	No significant change	No significant change
IV. Excretory	No significant change	No significant change
VI. Alimentary tract	Decrease	Decrease
VII. Nervous system	Decrease	Decrease

(3) The rates of change in time of the mortality are not significantly different in the urban and rural population moieties, so far as the organ systems here studied are concerned.

(4) There is a high degree of positive correlation between the synchronous deviations of the mortality rates from their trends in the urban and rural populations, in respect of each of the organological rubrics studied. The temporal factors causing variations in the values of the death rates from time to time during the period appear to have been operating simultaneously and congruently in both urban and rural areas.

SECULAR CHANGES IN DEATH RATES IN THE REGISTRATION STATES OF 1911 (WHITE POPULATION) AND IN ENGLAND AND WALES

The mortality data for the white population of the U. S. Registration States as constituted in 1911, and England and Wales, are particularly suitable for comparison. By taking the Registration Area as constituted at one particular time, any possible effects due to the changing size and composition of the U. S. Registration Area are eliminated. The two countries are made more comparable racially by the restriction of the material to the white population in the United States. Both countries long ago adopted the International Classification of the Causes of Death.

The underlying conditions affecting death rates in the two countries are of course not identical. The Registration States of 1911 and England and Wales differ from each other in climate and physical characteristics. The populations differ in their racial constitution. The general sanitary schemes and methods of public health administration of each country are peculiarly adapted to its own circumstances. Ways of living are also dissimilar.

The methods of analyzing time changes in the death rates assignable to the several organ systems in the U. S. Registration States of 1911 and England and Wales, during the period 1911-1922, are the same as those

used in the preceding section of this paper for the urban and rural portions of the U. S. Registration Area. The various organological classes are considered in the order of their appearance in the original classification. The data necessary for the discussion are presented in the Tables 7-11 inclusive.

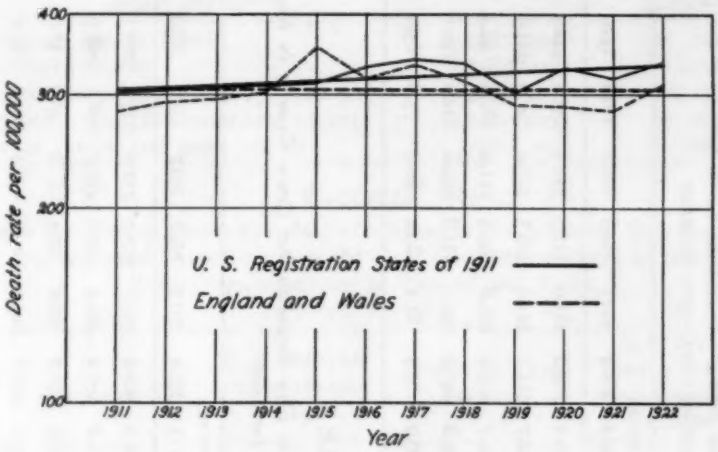

FIG. 7. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS I (CIRCULATORY SYSTEM, BLOOD AND ASSOCIATED ORGANS) IN THE U. S. REGISTRATION STATES OF 1911 (WHITE POPULATION) AND ENGLAND AND WALES, DURING THE PERIOD 1911-1922 INCLUSIVE

The mortality assignable to the *Circulatory system* is somewhat, though not greatly, higher in the United States than in England and Wales, with the exception of one year, 1915. The ratio of the two sets of mortality figures, United States/England and Wales, is $1.076 \pm .046$, which does not differ significantly from unity. The United States rates show a slight tendency toward increase during the period, which is expressed in the annual percentage rate of change $+ .761 \pm .212$. In England and Wales, on the other hand, no such tendency is apparent. The trend line is practically parallel to the abscissal axis, and the annual percentage rate of change is only $+ .017 \pm .418$, an obviously insignificant figure.

The deviations from the trends in the two curves are for the most part identical in direction at each point of time, but dissimilar in magnitude. The correlation coefficient for the synchronous deviations is $r = + .534 \pm .152$. This is probably a statistically significant value.

TABLE 7

Showing the Crude Annual Death Rates per 100,000 Living, for Five Organological Cause Groups in the U. S. Registration States of 1911 (White Population) 1911-1922 Inclusive

CLASS NO.	ORGAN SYSTEM	YEARS 1911	1912	1913	1914	1915	1916	1917	1918	1919	1920	1921	1922
I	Circulatory system, blood	303.7	304.0	309.2	309.6	313.0	331.3	339.6	335.4	302.4	330.0	319.0	337.5
II	Respiratory system	338.5	320.3	322.7	314.1	321.0	338.5	347.4	767.6	371.9	361.1	233.8	253.6
IV	Kidneys and related excretory organs	98.9	104.2	104.7	105.2	106.8	109.9	111.9	102.0	92.4	94.9	89.8	93.9
VI	Alimentary tract and associated organs	266.6	254.7	265.8	245.9	230.2	235.8	229.8	222.2	193.8	195.8	196.1	181.6
VII	Nervous system and sense organs	93.0	89.6	89.9	85.5	81.1	89.2	80.4	75.8	65.9	63.1	66.5	64.3

TABLE 8

Showing the Crude Annual Death Rates per 100,000 Living, for Five Organological Cause Groups, in England and Wales, 1911-1922 Inclusive

CLASS NO.	ORGAN SYSTEM	YEARS 1911	1912	1913	1914	1915	1916	1917	1918	1919	1920	1921	1922
I	Circulatory system, blood	282.3	292.3	296.2	302.6	355.6	317.0	332.9	314.1	288.1	287.5	284.0	311.0
II	Respiratory system	381.4	383.1	371.2	395.5	486.9	427.2	431.7	798.5	488.9	359.6	334.6	405.8
IV	Kidneys and related excretory organs	45.2	45.2	46.8	47.7	51.0	47.4	47.4	43.9	38.9	38.0	37.5	38.3
VI	Alimentary tract and associated organs	318.1	203.0	250.6	243.6	240.3	221.0	218.3	208.6	182.2	190.6	202.0	170.4
VII	Nervous system and sense organs	117.3	105.9	106.7	105.4	114.2	106.4	105.6	99.1	85.4	83.1	78.5	76.2

TABLE 9

Showing the Ratios of the Death Rate Curves for Five Organological Cause Groups in the U. S. Registration States of 1911 (White Population) and England and Wales, 1915 Values

CLASS NO.	ORGAN SYSTEM	RATIO UNITED STATES / ENGLAND AND WALES	ANNUAL PERCENTAGE OF CHANGE OF RATIO
I	Circulatory system, blood	1.076 ± .046	+ .743 ± .471
II	Respiratory system	.746 ± .062	— 1.459 ± 1.040
IV	Kidneys and related excretory organs	2.398 ± .055	+ 1.078 ± .540
VI	Alimentary tract and associated organs	1.041 ± .069	+ .239 ± .620
VII	Nervous system and sense organs	.796 ± .033	— .060 ± .460

TABLE 10

Showing the Annual Percentage Rate of Change in the Mortality Rate for Five Organological Cause Groups in the U. S. Registration States of 1911 (White Population) and in England and Wales, 1911-1922 Inclusive

CLASS NO.	ORGAN SYSTEM	U. S. REGISTRATION STATES OF 1911	ENGLAND AND WALES	DIFFERENCE UNITED STATES —ENGLAND AND WALES
I	Circulatory system, blood	+ .761 ± .212	+ .017 ± .418	+ .743 ± .469
II	Respiratory system	— 1.548 ± .766	— .090 ± .712	— 1.458 ± 1.046
IV	Kidneys and related excretory organs	— 1.170 ± .337	— 2.223 ± .403	+ 1.054 ± .525
VI	Alimentary tract and associated organs	— 3.447 ± .193	— 3.677 ± .563	+ .230 ± .595
VII	Nervous system and sense organs	— 3.720 ± .246	— 3.662 ± .369	— .058 ± .443

TABLE 11

Showing the Coefficients of Correlation of the Logarithms of the Synchronous Deviations of the Observed and Calculated Death Rates Assignable to the Same Organ System in Different Populations, for Five Organological Cause Groups, in the U. S. Registration States of 1911 (White Population) and England and Wales 1911-1922 Inclusive

CLASS NO.	ORGAN SYSTEM	COEFFICIENTS OF CORRELATION
I	Circulatory system, blood	+ .534 ± .152
II	Respiratory system	+ .494 ± .170
IV	Kidneys and related excretory organs	+ .886 ± .046
VI	Alimentary tract and associated organs	+ .330 ± .190
VII	Nervous system and sense organs	+ .656 ± .122

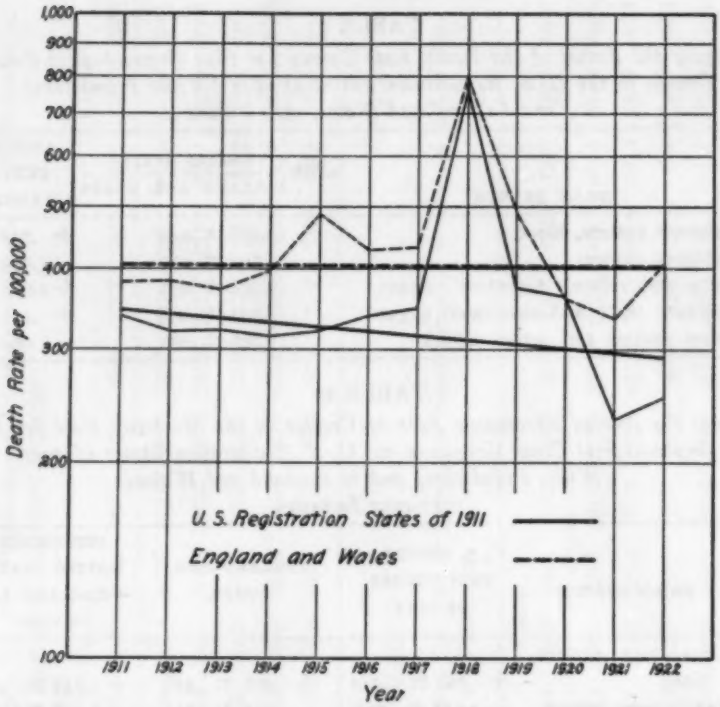

FIG. 8. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS II (RESPIRATORY SYSTEM) IN THE U. S. REGISTRATION STATES OF 1911 (WHITE POPULATION) AND ENGLAND AND WALES, DURING THE PERIOD 1911-1922 INCLUSIVE

Mortality assignable to the *Respiratory system* is much higher in England and Wales than in the United States. The ratio of the two respiratory curves, United States/England and Wales, is $.746 \pm .062$. The trend line of the English rates is practically parallel with the base, the annual rate of change being only $— .090 \pm .712$. In the United States the trend of mortality in this class was downward in the period studied, the annual percentage rate of change being $— 1.548 \pm .766$. The probable error of the percentage is so great, however, that the slope cannot be regarded as significant. The correlation between the synchronous deviations from the trend lines in the two curves is only $r = + .494 \pm .170$, a statistically insignificant value. If 1918 had been included in the computation, as perhaps it might fairly have been in this matter of correlation, the coefficient would have been higher.

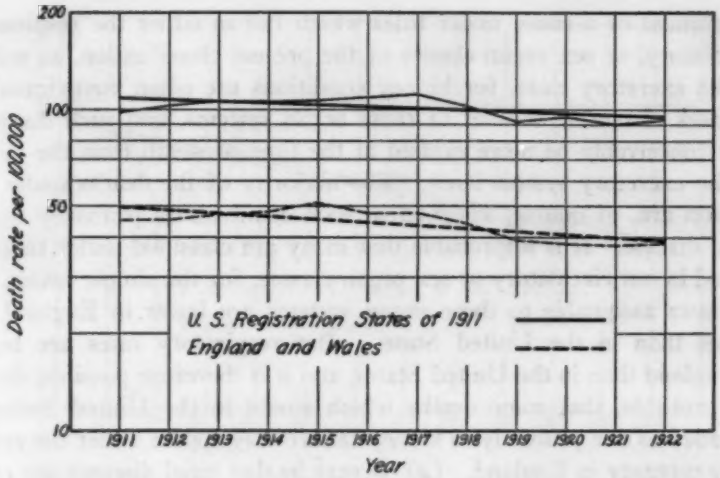

FIG. 9. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS IV (KIDNEYS AND RELATED EXCRETORY ORGANS) IN THE U. S. REGISTRATION STATES OF 1911 (WHITE POPULATION) AND ENGLAND AND WALES DURING THE PERIOD 1911-1922 INCLUSIVE

The comparison of mortality rates assignable to the *Kidneys and associated excretory organs* presents a puzzling problem. The United States rates are, on the average, more than twice those of England and Wales. The ratio, United States/England and Wales, is $2.398 \pm .055$. The rates of both countries definitely decrease with the passage of time during the period studied. The annual percentage rate of decline in the United States mortality figures is $-1.170 \pm .337$, and $-2.223 \pm .403$ in those for England and Wales. But the difference between the two countries in percentage rate of decline is insignificant in comparison with its probable error.

Two different suggestions may be offered in possible explanation of the great difference between the mortality assignable to the excretory system in the two countries. These are: (1) A certain number of deaths which would have been considered in the United States as primarily due to failure of the kidneys may have been recorded under other causes in England and Wales. Although the two countries have essentially the same system of tabulating mortality data, and although the directions for the assignment of joint causes are identical in the two countries, the decision as to the primary cause is made by the physician in filling out the death certificate. This offers opportunity for the

assignment of a cause under titles which fall in either the respiratory, circulatory, or sex organ classes of the present classification, as well as in the excretory class, for kidney conditions are often complicated by diseases of one or another of these organ systems, and such disorders may conceivably be more evident at the time of death than the failure of the excretory system itself. The majority of the deaths under discussion are, of course, assigned in both countries as primarily due to renal disease. It is improbable that many are classified under titles included in our circulatory or sex organ classes, for the simple reason that the rates assignable to these organ systems are lower in England and Wales than in the United States. But respiratory rates are higher in England than in the United States, and it is therefore possible, though not probable, that some deaths which would in the United States be regarded as due primarily to kidney failure may appear under the respiratory category in England. (2) It may be that renal diseases are *really* a relatively more frequent cause of death in the United States than in England. If so, the difference in the rates of the two countries may be due in some part to differences in the eating habits of the two populations. The diet of the American people apparently normally includes a greater proportion of protein than does that of the English people. For protein comprised 6.2 per cent of the total food consumed in the United Kingdom during the five year period 1909-1914 (6, p. 145), as compared with 10.2 per cent in the United States during the period 1911-1912 to 1916-1917 (3, p. 388-391). This may possibly be a factor underlying the higher excretory rates of the United States.

The coefficient of correlation of the synchronous deviations of the two series of kidney rates indicates a high degree of positive association. The coefficient is $r = + .886 \pm .046$. The general course of the two curves during the period is similar. In the early years of the time period, rates were slightly increasing in both countries. Following 1917 both sets of values show a decline.

In respect of the mortality assignable to Class VI, the *Alimentary tract and associated organs concerned in metabolism,* the United States rates are slightly above the English in every year except 1911, when there was an unusually high mortality from diarrhea and enteritis in England and Wales, and 1915 and 1921. The ratio, United States/England and Wales, is $1.041 \pm .069$, which does not differ significantly from unity, and shows no significant change in value during the period. The higher rates for Class VI found in the United States are generally

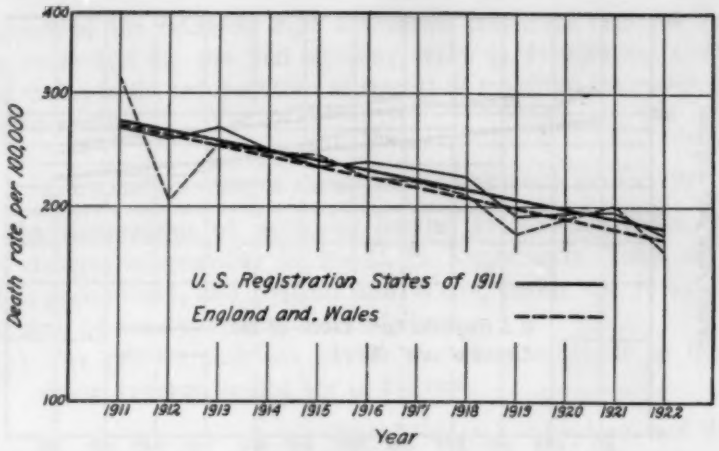

FIG. 10. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS VI (ALIMENTARY TRACT AND ASSOCIATED ORGANS) IN THE U. S. REGISTRATION STATES OF 1911 (WHITE POPULATION) AND ENGLAND AND WALES, DURING THE PERIOD 1911-1922 INCLUSIVE

due to greater mortality from diarrhea and enteritis in this country. The curves for both countries have a marked downward trend. In the United States the annual percentage rate of decrease is $-3.447 \pm .193$. That of England and Wales is $-3.677 \pm .563$. There is no significant difference between the two rates of decrease. Both trends are due primarily to the diminishing mortality from diarrhea and enteritis in childhood. The recorded mortality from cancer of the several organs included in Class VI increased during the period.

Factors affecting these death rates in the two countries did not result in deviations either identical in direction from the trends, or proportional in value at the same point of time. The correlation coefficient is only $r = +.330 \pm .190$. No significant association is shown between the synchronous deviations from the trend.

During the period studied the mortality assignable to Class VII, the *Nervous system and sense organs,* was heavier in England and Wales than in the United States. The ratio of the mortality rates, United States/England and Wales, is $.796 \pm .033$. The difference is primarily due to the higher rates from convulsions of infants in England. Both curves show a marked downward trend. The rates of the United States decreased $-3.720 \pm .246$ per cent annually; those of England and Wales by $-3.662 \pm .369$. These are well marked decreases with prac-

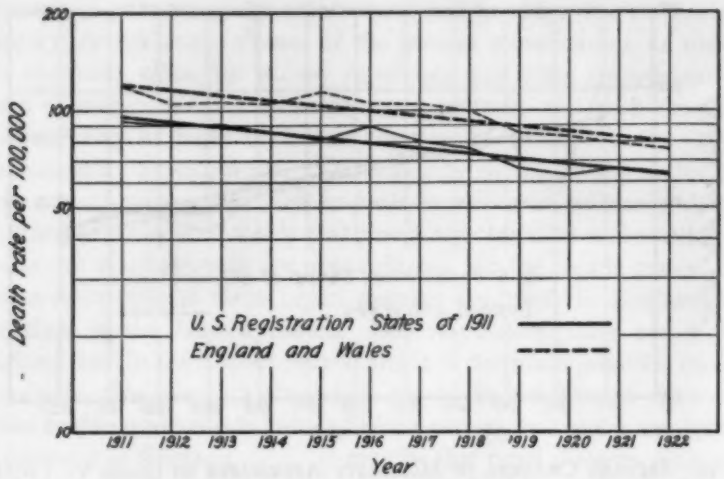

FIG. 11. SECULAR CHANGES IN MORTALITY ASSIGNABLE TO CLASS VII (NERVOUS SYSTEM AND SENSE ORGANS) IN THE U. S. REGISTRATION STATES OF 1911 (WHITE POPULATION) AND ENGLAND AND WALES, DURING THE PERIOD 1911-1922 INCLUSIVE

tically no difference between them. They are not, in either country, due to decrease from any single cause, but rather from declines in the rates of almost every title included in Class VII.

The deviations from the trend lines are of some interest. Prior to 1914 in England and Wales the mortality is below the fitted trend line, but from 1915 until 1918 inclusive the observed rates are above the trend line. In the last four years of the period under consideration the rates again dropped below the line. Prior to 1916 in the United States the rate is above the trend line in only one year. In the year 1916 we experienced a marked epidemic of poliomyelitis, and the highest United States rate of the whole period is observed. In the next two years, 1917 and 1918, rates continued higher than the normal values. In 1919 and 1920 the deviations went below the norm, and did not thereafter go significantly above it during the period studied. Correlation of the synchronous deviations gives the coefficient $r = + .656 \pm .122$, which is a statistically significant figure, indicating the action of temporary factors in both countries in a similar sense at the same points of time. In general, within the period covered in this study, low rates were found in both countries from diseases of the nervous system in the years of peace and high rates during the years of war strain. But the

existence of the relatively high correlation coefficient does not in any way prove that the war had anything really to do with the similarity of variation in the two countries in respect of mortality assignable to the nervous system.

SUMMARY REGARDING AMERICAN AND BRITISH MORTALITY

The observations of particular interest in the comparison of the time changes in mortality in the U. S. Registration States of 1911 (white population), and England and Wales, during the period 1911-1922 may be briefly summarized as follows:

(1) The relative positions of the mortality assignable to the five organ systems studied are as follows:

	United States	*England and Wales*
I. Circulatory	Higher	Lower
II. Respiratory	Lower	Higher
IV. Kidneys	Higher	Lower
VI. Alimentary tract	Higher	Lower
VII. Nervous system	Lower	Higher

However, the ratios for the Circulatory system and the Alimentary tract do not differ significantly from unity.

The ratios between the mortality of the two countries show no significant tendency toward change with time during the period studied, so far as concerns the five organ systems considered.

(2) Evidence of secular change in the rates of mortality assignable to the several organ systems during the period studied is as follows:

	United States	*England and Wales*
I. Circulatory	Increase	No significant change
II. Respiratory	No significant change	No significant change
IV. Kidneys	Decrease	Decrease
VI. Alimentary tract	Decrease	Decrease
VII. Nervous system	Decrease	Decrease

(3) Correlation of the synchronous deviations from the trend lines in England and Wales and the United States indicates a significant degree of association for the following organological classes: I. Circulatory system, IV. Excretory system, and VII. Nervous system. In all cases the correlation is positive in sign.

THE MUTUAL INTERCORRELATION OF THE MORTALITY ASSIGNABLE TO THE MORE IMPORTANT ORGAN SYSTEMS

So far in this study of secular changes in mortality we have set forth the general characteristics of the mortality assignable to each organ system in each population, with reference to rate of time change in rates, and we have analyzed the correlation between synchronous deviations from the trend in the mortality assignable to some organ system in pairs of compared populations (Urban and Rural, and American and British).

The next step is to examine the correlations between the synchronous annual fluctuations from the trend lines in (a) the mortality assignable to one organ system, as, for example the circulatory, and (b) that assignable to a different organ system, for example the respiratory, *in the same population*. These correlations have been computed, in all possible pairings, for the first four of the five organ systems here discussed. The results are given in Table 12.

From Table 12 the following observations may be made:

1. The correlations between the deviations from the trend lines for mortality assignable to the circulatory and respiratory systems are all negative in sign and insignificant in value (having regard to the probable errors) in all the United States population groups, for the time period studied. In the English population, however, the coefficient is positive in sign, and more than four times its probable error. This marked difference between American and English populations seems inexplicable on any biological grounds. More probably it arises from differences in nosological, diagnostic, or registration points of view in the two countries.

2. The circulatory *cum* excretory system correlations are all positive in sign and high in value. This is a reasonable and expected result, in view of the known close relationship, physiologically and pathologically, between these two organ systems.

3. In the United States populations the circulatory *cum* alimentary system correlations are consistently positive and relatively high in value. Again, however, there is a marked departure of the English population. There the coefficient is zero, within the error of sampling. The United States values seem reasonable biologically, but no explanation is apparent for the English finding. It may be that the years chosen made a particularly deviant sample. Or, again, it may be in some obscure way a result of differing registration practice.

TABLE 12

Showing the Coefficients of Correlation of the Logarithms of the Synchronous Deviations of the Observed and Calculated Death Rates Assignable to Four Organological Cause Groups in the Same Population

CLASSES CORRELATED	ORGAN SYSTEMS CORRELATED	REGISTRATION AREA, U. S. A.		U. S. REGISTRATION STATES OF 1911 (WHITES)	ENGLAND AND WALES
		Urban	Rural		
I and II	Circulatory and respiratory	− .033 ± .238	− .477 ± .191	− .066 ± .224	+ .640 ± .133
I and IV	Circulatory and kidney	+ .673 ± .123	+ .792 ± .084	+ .782 ± .083	+ .944 ± .023
I and VI	Circulatory and alimentary tract	+ .811 ± .077	+ .682 ± .120	+ .664 ± .119	+ .132 ± .213
II and IV	Respiratory and kidney	− .501 ± .179	− .730 ± .111	+ .236 ± .212	+ .547 ± .158
II and VI	Respiratory and alimentary tract	+ .380 ± .204	− .112 ± .235	− .239 ± .212	− .403 ± .188
IV and VI	Kidney and alimentary tract	+ .367 ± .195	+ .462 ± .177	+ .536 ± .152	+ .119 ± .210

4. The respiratory *cum* excretory results are rather contradictory, and permit of no clear interpretation.

5. There is apparently a tendency for the respiratory *cum* alimentary system correlations to be negative in sign (with the U. S. Urban popu-

lation alone out of line) but they are all statistically insignificant, having regard to their probable errors.

6. The excretory *cum* alimentary system correlations are consistently positive in sign, but low in value, having regard to their probable errors.

SUMMARY AND DISCUSSION

This study is concerned with the changes in mortality assignable to five organ systems (Circulatory, Respiratory, Excretory, Alimentary, and Nervous) according to Pearl's organological system of classification, during a time period from 1910 to 1922, and in four different population groups, viz.: Urban portions of U. S. Registration Area; Rural portion of U. S. Registration Area; U. S. Registration States of 1911 (white population); and England and Wales.

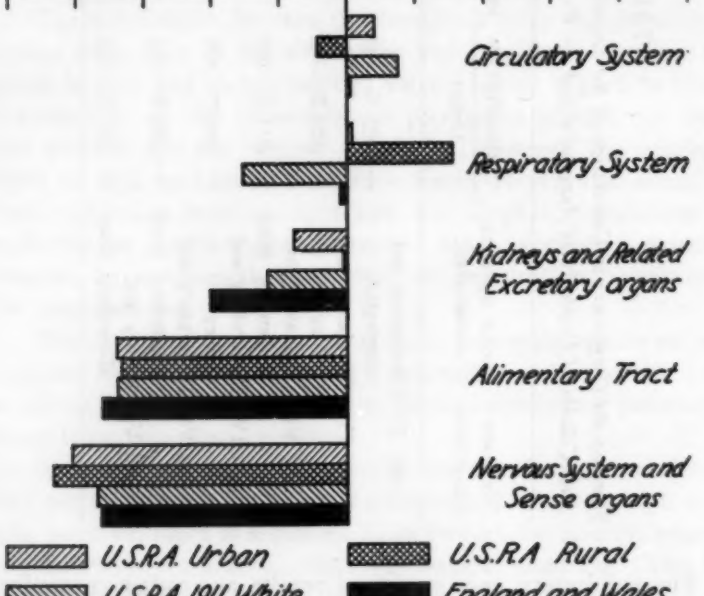

FIG. 12. ANNUAL PERCENTAGE RATES OF CHANGE IN THE MORTALITY ASSIGNABLE TO FIVE ORGANOLOGICAL CAUSE GROUPS IN FOUR POPULATION GROUPS

The most important general result of the study is shown graphically in Fig. 12.

Broadly speaking, Fig. 12 shows that during the time period studied there was no consistent or important trend change in the mortality assignable to the Circulatory or Respiratory systems, due allowance having been made in the computations for the 1918 influenza pandemic. There was a consistent but not strongly marked downward trend in the mortality assignable to the Excretory system. The mortality assignable to the Alimentary and Nervous systems declined markedly and consistently during the period, in all four population groups.

There were found to be rather unexpectedly high correlations between the urban and rural populations of the United States, and also between the American and British populations, in respect of the synchronous deviations of the mortality rates of all the five organ systems from the respective trend lines. When the Circulatory mortality went up in the United States it also went up in England, and when it fell below in one of these countries it also fell below in the other.

The results of this study are, by and large, consistent among themselves. They seem simple and straightforward. But they are extremely difficult to interpret in any proper scientific sense. Annual rates of decline of mortality of from 4 to 5 per cent, such as are seen in the Alimentary and Nervous systems, represent substantial movements. The phenomena to be interpreted are, in short, real. They require no fine apparatus of measurement to be detected. But what caused them? The simple and obvious answer for the Alimentary mortality is that it was ever-improving public health and sanitary measures. Very likely so. But if this is the explanation, it does seem odd that the rate of decline should have been so precisely similar in the urban and rural populations of the U. S. Registration Area. There would seem to be no semblance of a scientific reason to allege that the marked decline in the mortality assignable to the Nervous system was due to health efforts. Probably no one would allege it. Yet there it is, in a period which included the World War, and the decline in England was practically as great as in the United States.

It seems to us that such observations as are recorded in the paper clearly point to, and emphasize, the necessity for a great deal more, and more penetrating, research than has yet been done before it will be possible to reach any adequate understanding of the secular changes which are going on in human mortality rates.

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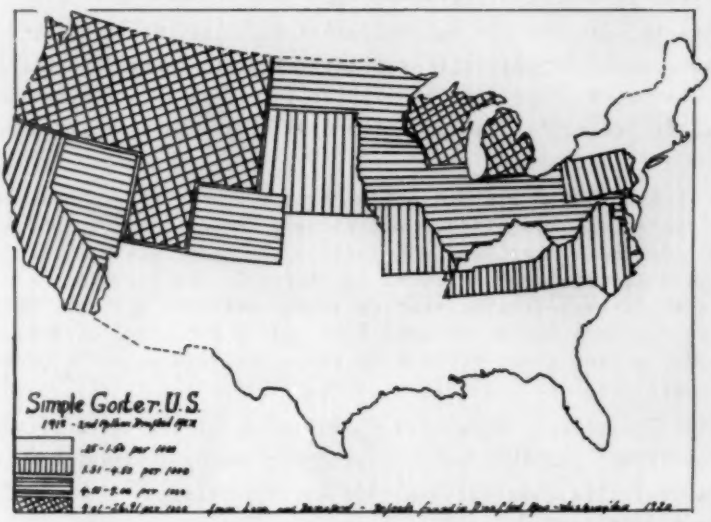
SOME EFFECTS OF THYROID DISORDER IN WOMEN

BY L. S. CRESSMAN

Department of Sociology, University of Oregon

The purpose of this study[1] is to discover whether any significant differences exist between a group of women with thyroid disorder and one in which the gland functions normally; and if there are differences what they may be.

The data consist of the health records of 375 women students of the University of Oregon of the class of 1933-34. We excluded 29 cases of students from California, the Hawaiian Islands and other places outside of the "goiter belt"[2] in order to isolate the possibility of other environmental factors than thyroid disorder influencing our results. Our



[1] I am particularly indebted to two of my advanced students, Miss Katherine Bluhm and Miss Edna Spenker, for their able assistance in making this study.

[2] Cf. Map of Distribution of Simple Goiter in the United States.

results, therefore apply to 375 and not the entire group of women students. There were 296 or 78.9 per cent which showed a normally functioning thyroid and 79 or 21.1 per cent an abnormal gland.

Our records do not distinguish between hyperthyroidism and hypothyroidism—a distinction eminently desirable—for in the final analysis this difference can be definitely determined only by the basal metabolism test. Heart action and respiration are factors which may be used in a diagnosis but they lose their usefulness in connection with these health records because most of the students are in such a state of excitement during the first week of college that they show records on these tests that are not reliable. The ratio of endemic goiter to exophthalmic in Oregon as shown by the Surgeon-general's report [3] is about one in five or six. If the characteristics of these two types tend to cancel out each other as clinical evidence indicates, we shall expect that our results show the differences characteristic of endemic goiter to only a limited degree. In other words, whatever our results may be, any differences caused by endemic goiter will appear to be smaller than they actually are because of the inclusion of the exophthalmic group with the endemic.

The question arises, in this connection, of the reliability of the physical examination for abnormal or enlarged thyroid. The system used at the University is that customarily followed in which the examiner stands behind the subject.[4] The method and its effectiveness is described as follows:

> Dr. J. Earl Else, of Portland, Oregon, says, in a personal communication: "I am of the opinion that by the use of the method developed in this clinic we can palpate all thyroids except those with a retrotrachael development. This method consists of standing behind the patient and placing the first three fingers of each hand over the thyroid region while the patient swallows. I regard the small palpable thyroid as normal when the lower pole is not blunt. A blunt lower pole either means a goiter present at the time of examination, or the remains of a previous goiter . . ."[5]

The University's method of classification of the results follows: "not palpable; palpable but not enlarged; enlarged; nodular; toxic symptoms." We classified "palpable but not enlarged" with the "non-palpable" as normal thyroids. This may be leaning over backwards and

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[3] Love, Albert G., M. D., and Davenport, Charles B., "Defects Found in Drafted Men," War Dept., Washington, D. C., 1920, pp. 110-111.

[4] Card showing information used in study.

[5] Oleson, Robert, Surgeon U. S. Public Health Service. Endemic Goiter in Oregon. Reprint No. 1189 from the *Public Health Reports*, Nov. 18, 1927 (Pages 2531-2849), p. 4.

admits the possibility of omission of abnormal thyroids classed in our normal group. However, any other classification is impossible unless the other group is unduly weighted. Either way differences between the groups due to thyroid deficiency would tend to be eliminated. The data card used for this study follows:

Number Age Yrs. Mo. Home address
Ht. Wt. Overwt.
Breasts—Small Moderate Large Masses Retracted nipples

MENSTRUAL HISTORY

Age began Amount—Scanty Moderate Profuse
Pain—Mild Moderate Severe Occasional Frequent Always No. of days

THYROID

Not palpable; palpable but not enlarged; enlarged; nodular; toxic symptoms
Headaches—often occasional never (Nervous Breakdown?)

	Extreme 1	Moderate 2	No
1. Inclined to worry			
2. Often discontented			
3. Feelings easily hurt (sensitive)			
4. Deliberate in making decisions			
5. Prefer to be alone			
6. Dislike to be alone			
7. Fond of day dreaming			
8. Nervous when in danger			
9. Always cautious about everything			
10. Find it difficult to speak in public			
11. Cautious about making friends			
12. Moods change readily			
13. Have been troubled by a sex problem			
14. Blush easily and often			
15. Easily discouraged			
16. Easily irritated			
17. Often have the blues			
18. Troubled with shyness			
19. Have had trouble with other people			
20. Dislike company of girls			
21. Dislike company of boys			
22. Subject to fainting spells			
23. Nervous			
24. Like to lead			
25. Like to follow			
26. Have spells of uncontrollable anger			
27. Troubled by disagreeable dreams			

28. Walk in sleep
29. Have dizzy spells
30. Have been troubled by stuttering
31. Inclined to laugh too easily
32. Have "hysterics" occasionally
33. Cry too easily

The main function of the thyroid is to control metabolism. Consequently, if thyroid disorder develops it should result in symptoms of disturbed physical growth or functioning. This gives us, then, the possibility of disorders resulting from generally disturbed metabolism. Then physical changes or disorders may be reflected in the psychic and the mental life of the individual. Furthermore, there is the possibility of a direct thyroid-ovarian relationship as indicated by Harrow, although Hammett's experiments tend to disprove this. However, we are not sure that what is true for rats is true for humans in this connection, especially since Hammett found that in rats "the reproductive system of the male reaches maturity at an earlier age than does that of the female" in direct contrast to humans.[8] These possibilities of disordered functioning lead to three possible groupings of results:

I. The Physical Characteristics
II. Psychological or Psychic Traits
III. The Academic Ratings

I. The physical characteristics will include:

a. The height-weight ratios
b. The menstrual history. This will consider:

1. The age of pubescence or the age of the first menstruation
2. Inter-menstrual period
3. Nature of menstrual flow, i. e., amount
4. Presence of pain during menstruation
5. Number of days pain
6. Character of pain with respect to severity
7. Development of the breasts—

Menstruation and mammary development have both been shown to be related to the secretion of the ovarian hormone, which in turn may be affected by the thyroid secretion

8. Occurrence of headaches.

[8] Hammett, Fred. S., Studies of the Thyroid Apparatus, XXXIII. *Am. Jour. Physiology*, Vol. 77, pp. 527-546.

II. The psychological or psychical characteristics consider

1. The comparison of the two groups for individualistic and group behavior characteristics.

III. The academic ratings. These are comparisons of the group

1. By the median percentile rating
2. By the mean point-grade score for the winter term 1929-1930
3. Academic mortality at the end of Fall Quarter 1930-1931, i. e., after four quarters.

It is fairly obvious why girls should be chosen for this study. The arrival of puberty or pubescence in girls is marked by the onset of menstruation.

THE ANALYSIS OF THE DATA: PHYSICAL CHARACTERISTICS

The human body under normal conditions shows a definite relationship between height and weight. This of course varies between the sexes, races and family trees within races. However, the relationship is such that the average height for any group and the variability of the units has been established. We are thus able to make three different sets of comparisons.

1. Height and weight
2. a. Height with reference to variability
   b. Weight with reference to variability
3. The correlation of height and weight for the two groups.

Obviously it would be better if we had sufficient observations to make our comparisons by age classes of single year groupings. But we are not prevented from using the correlation or comparisons because all our subjects are in the upper age groups of post pubescent or early mature development. This is further indicated when the comparison of our "*r*" is made with a similar index from an English study where correlations of growing children are worked out by single weeks of age. These "*r*'s" range from $.694 \pm .059$ to $.827 \pm .032$ for Edinburgh.[1] Our "*r*" for the normal group is $r = .771 \pm .02$. Obviously we can use our group as a whole.

[1] Child Life Investigations, Poverty, Nutrition and Growth, Special Report Series, No. 101, p. 33. H. M. S. Office, London, 1926.

Height

The median height for the normal group is 63.92 inches, while that for the abnormal is 64.03 inches, a difference of but .11 inches in favor of the abnormal group.

The mean, which is a measure of magnitudes rather than position, is a better index of comparison in this case. The mean height ($M \pm \sigma$) of the normal group is 63.88 ± 2.26 while for the abnormal group it is 63.79 ± 2.30 inches. The difference of the means should be fully three times as large as the P. E. difference of the means to be significant. But here we find that the P. E. difference M_1M_2 is .09. Obviously the difference is not significant.

If thyroid disorder affects metabolism then there should be a greater variability in the height of the abnormal group than in the normal. The coefficient of variability is a measure of this factor and the coefficients for two groups may be compared. The coefficient of variability for the normal group is 35.4, while that for the abnormal is 36.1. Thus, while there is a negligible difference between the groups as measured by the median and mean there is shown to be a greater variability in the abnormal group, to some extent indicative of the disturbed metabolism in this group.

Weight

The body weight should be a better index of thyroid disorder than height. While maximum height may be pretty well reached at the completion of puberty the height-weight ratio will not remain fixed because if the metabolism of the body is affected it will of necessity have to affect primarily weight. The development of thyroid disorder increases after puberty in girls as has been shown for Oregon.[8] Consequently weight would be more likely to be affected than height and be more reliable as an index. What do we find?

The median weight for the normal group is 121.01 pounds, and for the abnormal group slightly less, 119.50 pounds, an almost negligible difference. But the median is a position average and takes no consideration of the magnitudes. When we consider the mean we find that for the normal group the $M_1 \pm \sigma = 121.66 \pm 16.87$ pounds, while for

[8] Oleson, Robert, U. S. Public Health Service, Reprint No. 1189, Nov. 18, 1927. p. 11.

the abnormal it is $M_2 \pm \sigma = 122.97 \pm 20.52$ pounds. The probable error of the difference of M_1 and M_2 is but slightly greater than the difference of the means, which would indicate again a lack of significance. However, there is a contrast between these measurements and those for heights, that is, while the mean height of the normal group was slightly greater than that for the abnormal, in the case of weights we find the difference reversed, whereas the normal situation would have maintained the relative relationship.

This gradually developing difference is further brought out where we compare the coefficients of variability for the groups. We find coefficient of variability for the normal group is 13.8, while for the abnormal it rises to 16.6. In other words, the abnormal thyroid group shows greater variability in weight than the normal thyroid.

If body weight has a "tendency to increase" with endemic goiter then we should consider the proportions of each group above and below normal weight and the distribution of each of these classes. We find as a matter of fact that the percentages are almost the same for the two groups. The normal has 25.3 per cent under weight, the abnormal 27.8 per cent; the normal has 24.7 per cent overweight, the abnormal 22.8 per cent. The normal group thus has 50.0 per cent reckoned as normal and the abnormal 49.4 per cent. These percentages are too crude as measurements, for what we want to know finally is: How do the two groups compare with reference to the distribution of the underweight and the overweight classes? We should expect differences here in view of the higher coefficients of variability for the abnormal group.

As a matter of fact we find the median of the underweight series for the normal group to be 14.50 pounds, and for the abnormal 14.55, certainly an insignificant difference. The series which should show a difference if the hypothesis is correct is the overweight group. Here we find the median for the normal overweight series is 15.45 pounds, while that for the abnormal is 20.50 pounds. This would seem to show conclusively that there is a general tendency to put on weight as a characteristic of the group, especially in a few cases, extreme weight. But it would still seem that the differences must be based upon the greater variability of the abnormal group, considering, of course, the general tendency for the abnormal observations to be skewed toward the top of the curve.

A still further test of this symptom of disturbed metabolism and probably the most significant is the coefficient of correlation of the two

variables, height and weight. We find that $r \pm$ P. E. for the normal group is .771 ± .02, while for the abnormal it is .404 ± .09. This r for the normal group we already compared with those for normal children from Edinburgh and saw the correspondence. The fairly low $r = .404$ for the abnormal group is significant, but much lower, and indicates a much less close correspondence between height and weight than for the normal series.

We may summarize our height-weight findings by saying that there appears to be no significant difference between the two groups with regard to the mean height or mean weight. Half of the normal group are less than 63.92 inches in height, while the abnormal group shows half to be less than 64.03. Half of the normal group is under 121.01 pounds, and the abnormal shows half under 119.50, slightly lower than for the normal, although the median height was slightly greater. The variability for the abnormal group is greater for height, and weight as measured by the coefficient of variability and the coefficient of correlation, and this greater variability is especially brought out in the medians of the over-weight series.

Menstrual History

1. *Age of First Menstruation*

If Hammett's experiments with rats showing a retarded onset of puberty be significant for humans we should expect the age of first menstruation for the abnormal thyroid group to be later than that for the normal. The theory of the direct thyroid-ovarian stimulating relationship would require the same results to appear.

Our observations, however, show the following for the age of first menstruation for the two groups.

	Normal	*Abnormal*
Median	13.07 years	13.02 years
Mean ± σ	13.14 ± 1.25 years	13.05 ± 1.39 years
Mode	12.94 years	12.94 years
V	9.5 per cent	10.6 per cent

P. E. diff. $M_1M_2 = .11$

$M_1 - M_2 = .09$

None of these differences are statistically significant, nor would the differences which they represent in pubescence be socially significant, being a matter of about fifteen days.

Does this correspondence rule out the possibility of the retarding influence of thyroid deficiency upon pubescence? What factors are generally held to affect puberty? And may it possibly be the length of pubescence which is the significent factor? If there is no difference in pubescence for the groups then the possibility of developments of social significance resulting from that assumed difference disappear. Fortunately we have a check. Let us first consider what factors affect pubescence to discover if there may be factors which are perhaps stronger in our series than the thyroid disorder.

Plöss-Bartels in *Das Weib in Natur-und Völkerkunde* writes as follows on factors affecting the first menstruation: "Not alone through climate will the onset of menstruation be affected, but through many other relationships, for example, race, nationality, habits of life, occupation, breeding (education), locality, dwelling, clothing, manners and customs." A little further on we find,

> There are without doubt other factors to be considered which can be of no less limiting influence on the earlier or later onset of menstruation than those named earlier. Heredity is of the very first importance among these. We mean by this not the simple heredity of a nationality, but the so often neglected transmission of individual properties to the following generation. Thus one learns not infrequently at least among our population that the daughter first menstruates in exactly the same year as her mother and grandmother, and this conformity applied very often to the duration and the quantity of the elimination. Then, too, that which we have commonly called temperament, is certainly more to be considered than is commonly the case; by this is meant the characteristics of the development of the body.[9]

The final caution is that the whole milieu of the individual's life must be considered in arriving at the causes. Since our college students, and the women probably more than the men, represent a selected class, it may be that the factors involved are common in enough cases to produce the overlapping, assuming that the thyroid disorder should be a factor. At any rate we have insufficient observations to make any further correlations and yet have statistically significant results.

We do have another method of verifying our hypothesis or at least of securing some suggestive evidence in regard to it. Hammett has discovered in guinea pigs that

> The percentage rate of growth of the uterus is greater than that of the ovary save at the height of puberty, or at the period immediately succeeding the first appearance of corpora lutea. This shift is exhibited in both series.

[9] Plöss-Bartels, Das Weib, Nufeld und Henius, Berlin, 1927, p. 675.

Combining this fact with the fact that the post-pubertal drop in percentage rate of growth of the ovary precedes and is greater than that of the uterus it is clear that the development of the uterus lags behind that of the ovary, and that it is not completed during puberty when complete functional effectiveness of the ovary is attained.[10]

If there is a difference in ovarian and uterine maturation then the failure of uterine functioning in pregnancy should show (for early and later pregnancies) a differential death rate from puerperal albuminuria and convulsions or eclampsia, both being diseases which may be related to faulty functioning of the uterus. This early death rate from these diseases should be accompanied by a high rate for the late years of life, e. g., 40-50, as a result of organic deterioration. That there is this correspondence was shown in a study by Dr. Lee K. Frankel of the Metropolitan Life Insurance Company in a paper called "The Present Status of Maternal and Infant Hygiene in the United States." The death rates for these are highest in the years 10-19 and 40-49.[11] Following out our suggestions, if this assumption is correct, then the maternal death rate for this disease should be higher for mothers from 10-14 years than for those from 15-19 years of age. When we take our mortality statistics for states and by race the numbers of births to mothers under 15 becomes too small to be used. But if we consider the United States as a whole then we have a fairly good collection of data. We find that the maternal deaths from puerperal albuminuria and convulsions per 100,000 live births for the United States by race are as follows:

	1920	1923	1925	1927
		10-14 years of age		
White	534.8	419.0	941.2	595.2
Negro	5750.0	3021.6	2269.9	1948.6
		15-19 years of age		
White	304.7	283.8	289.4	202.1
Negro	1138.2	760.8	949.0	446.4

If diet is among the more important factors in bringing on these diseases because of the extra effort it puts upon metabolism and elimination, we should expect this variation between the negro and white, who represent such different social and economic conditions. It is also

[10] Hammett, *op. cit.*, p. 532.

[11] Frankel, Lee K., Ph. D. "The Present Status of Maternal and Infant Hygiene in the United States," New York, 1927, p. 2.

important for our point that the same type of difference holds between the age periods of the two races for the years considered.

The question is: What is the relation of goiter to maternal deaths from these causes? Therefore, we calculated the coefficient of correlation for incidence of goiter, using the army figures for men to rank the states (presumably there would be a comparable ratio for women) and the maternal death rates from these causes for women 10-19 years of age. We used the year 1920 as nearest to the time when the army records were taken and the first year after the influenza epidemic had spent its force. Using the rank difference method of computation especially applicable in this kind of relationship (ranking), we find $\rho = .520$, which when read as $r \pm$ P. E. $= .54 \pm .11$. We have then a positive correlation and a fairly high one with a small P. E.

The objection might be raised that this higher maternal mortality is due to the disturbed metabolism of the mother with thyroid disorder. The assumption entirely fails to account for the sudden drop in this death rate after 20, the low rate until 40 and then the resurgence, especially in view of the fact that thyroid disorder is accentuated during pregnancy and that thyroid disorder, we know, has higher incidence at 20 than at 15 or 18 years of age, unless of course, prophylaxis has been resorted to.

We must conclude then that our evidence tends to show that there is at least later uterine maturity for women suffering from thyroid disorder than for the normal group. As we suggested, the variability, characteristic of the other physical traits, may be reflected in the length of pubescent development. The other traits, looking ahead for a moment, all point to a retarded group, and there is no reason to believe that we have an exception here, and our evidence suggests certainly a retarded uterine maturity, which in its turn implies a retarded pubescence.

The anterior lobe of the pituitary may be more effective in the cases of reduced thyroid activity and so stimulate ovarian activity. Our r would tend to indicate that the pituitary effect would be an ovarian specific and not related to uterine maturity.

2. Inter-menstrual period

From the theory of the stimulating influence of the thyroid on the ovarian activity, one would expect to find a longer inter-menstrual period for the abnormal group. There is plenty of evidence for a rela-

tionship between heat in animals and the secretion of an ovarian hormone. There is also the evidence of the ovarian-thyroid relation. Hypothyroidism is a retarding factor and hyperthyroidism should be an accelerating influence. But we do not find the differences we should expect reflected in our data. We have

	Normal Group	*Abnormal Group*
	Days	Days
Mean	28.66	28.18
Median	28.16	28.12
Mode	27.16	28.00

On inspection none of these differences are significant. The abnormal group is lower in the mean and the median and slightly higher in the mode.

Why do we not find the expected differences? We can only speculate on that. First we do not have the hypothyroid separated from the hyperthyroid and for Oregon the Surgeon-general's report showed a ratio of about 1 in 5 to 6 cases of endemic goiter.

Second, as in the age of first menstruation, we undoubtedly have a complex of factors affecting the recurrence of menstruation. Plöss-Bartels referred especially to the influence of heredity upon the duration of the menses.

Third, we may have a different phenomenon to a great degree for humans when compared to other animals. Menstruation and heat are not the same thing functionally, either physiologically or in outward behavior. In humans the desire and ability to copulate and conceive is not limited to a menstrual period, but is a potentially constant thing as are the developed breasts and other secondary sexual characteristics, although Davis has recently shown the increase of sexual desire around the menstrual period. Hamilton and MacGowan write:

> At all other times these animals are sexless, unless we cite as exceptions that the females are mothers and, in many cases, enjoy certain immunities granted them by the males of the tribe.
>
> With primates it is different. The females are sexually responsive and sexually provocative at all times, except for a brief period of a few weeks or a month or two just before the birth of the young. She monkey, she baboon, she ape, and she man—they are all alike in refusing to lock up the most urgent and colorful of their emotions between the periods of physiological preparedness for conception.[12]

[12] Hamilton, C. V., and MacGowan, Kenneth, "What is Wrong With Marriage?" New York, Albert and Charles Boni, 1930, p. 9.

3. Amount of menstrual flow

Since this is measured by general terms of scanty, moderate, profuse, we are not in a position to give anything but a rough statement of the subject's own estimates, remembering the lack of exact definition of what they might mean.

	Scanty		*Moderate*		*Profuse*		*Not Recorded*		*Total*	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Normal	34	11.48	256	86.48	4	1.35	2	.76	296	100.
Abnormal	12	15.20	66	83.54	1	1.26	0	.00	79	100.

While the calculation of percentages when the base is less than 100 is a somewhat questionable procedure, here it is nearly 100 and so our percentages are not affected sufficiently to invalidate comparison with the larger group. We find here definite signs of a deficiency in the sexual function when measured against the norm. The abnormal group show 3.72 points more reporting a scanty flow and 2.94 points less moderate flow than for the normal group. The profuse group is slightly higher but not significantly so for the normal. Here again we find evidence of retardation and deficiency.

4. Presence of Pain

Measured according to its appearances as

	Never		*Occasional*		*Frequent*		*Always*		*Total*	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Normal	106	35.81	94	31.75	19	6.42	77	26.02	296	100.
Abnormal	25	31.64	24	30.38	6	7.60	24	30.38	79	100.

Here we find the advantage definitely in favor of the normal group. In the "never" group we find a difference of 4.17 points in favor of the normal group and in the "always" group we find 4.36 points in favor of the abnormal. Considering the presence of pain as an interfering factor in the work of the individual we would place the abnormal group at a disadvantage, especially since the "always" figures are supported although to a small degree by the "frequent" figures.

5. Quality of Menstrual Pain

Our classes here are none, mild, moderate, severe.

	None		*Mild*		*Moderate*		*Severe*		*Total*	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Normal	104	35.2	74	25.0	75	25.3	43	14.5	296	100.
Abnormal	25	31.7	25	31.7	14	17.6	15	19.0	79	100.

We find 3.5 points more of the normals are free from pain than the other group, while 6.65 points more of the abnormal have mild pain, 7.6 points more of the normal show moderate pain, and the abnormal exceed the normal in suffering severe pain by 4.5 points. Here again we have the fluctuations in the extremes for the abnormal group going beyond the normal in each case.

6. Number of days Pain

We have calculated the means in this case and find a very small difference. .87 days duration of pain for the normal and .91 days for the abnormal group. The difference is small but is cumulative when added to the greater percentage of the abnormals suffering pain always and the similar proportion experiencing severe pain.

Obviously we have a factor here in menstrual discomfort and deviant functioning which seems to be a result of thyroid disorder and which is of significance for personal adjustment as well as social adjustment.

7. Size of the Breasts

The breasts are likely to be more responsive to the stimulation of the female hormone than to the general growth process. Injection of the female secretion to which we have already referred shows this in its effect upon the mammary glands. Our data bear out the general results found so far in this field by clinical experience.

	Small		*Moderate*		*Large*		*Nothing Recorded*		*Total*	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Normal	116	39.2	148	50.0	27	9.1	5	1.7	296	100.
Abnormal	39	49.4	34	43.0	6	7.6	0	.0	79	100.

We have here again the difference which indicates the retarded nature of the abnormal group and indicates again the probable results of endocrine deficiency. This is especially striking in view of the greater weight of the abnormal group. If size of breasts were depend-

ent upon physical growth and weight we should expect just the opposite of what we find here. Small breasts result from thyroid deficiency. It would be interesting to follow this with reference to the capacity of mothers suffering from thyroid deficiency to nurse their children as compared with a normal group.

8. Occurrence of Headaches

These headaches may be of either an organic or functional nature. The records do not pretend to differentiate. Our data show:

	Never		*Occasional*		*Often*		*Nothing Recorded*		*Total*	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Normal	102	34.45	177	59.80	16	5.40	1	.35	296	100.
Abnormal	23	29.11	49	62.02	5	6.33	2	2.53	79	100.

Again we have the abnormal group at a disadvantage when considering personal and social adjustment. The normal group shows a superiority in the "never" group of 5.34 points, while the abnormal group has 2.22 per cent more having occasional headaches and 1.00 per cent more having headaches often. This is an interesting item in connection with study and other phases of academic adjustment.

Summarizing our results of this section we may say that for all points of comparison with regard to the menstrual history, with the possible exception of the recurrence of the menstrual period, the abnormal group is at a disadvantage when compared with the normal group using as our standard of measure deviation from a norm. In addition, when this deviation expresses itself in terms of extreme pain, recurrent headaches, longer experience of pain, we have specific points of difference showing the difficulty under which the abnormal group works when compared with the normal. This suggests, of course, the advisability of different regulations of absences from work for women than for men and different standards among the women for the normal and abnormal. That this is no pipe dream is suggested by the last part of our study, the academic ratings.

PSYCHIC CHARACTERISTICS

Our data included a list of personality traits which are calculated to indicate a tendency toward a socially non-participating or a socially inclined personality, which we shall call for convenience's sake, indi-

vidualistic or social. There are thirty-three items listed. These are checked by the examiner according to whether the subject states the existence of the trait, as extreme, moderate, no. For example, the first trait is "inclined to worry." It may be checked extreme / moderate / no. Of course, it is realized that these items are not exact. They are indicative of personal ratings and we may grant them some validity here.

We took the total number of times these items were scored by each group and then calculated the percentage each possibility was of the total number of items.

We find the following results:

	Extreme Per cent	*Moderate* Per cent	*None* Per cent
Normal	6.41	31.84	61.75
Abnormal	9.05	34.07	56.88

This would indicate that the abnormal thyroid group has a tendency to be individualistic, whereas the contrary is the case with the normal group.

ACADEMIC RATING

Academic ratings for the two groups were compared on the percentile rating scale and the grade point hour scale. The hours completed are too much affected by academic requirements to have any significance.

When compared on the psychological Percentile Rating [18] scale we find that the median, the significant measure here, is .411 for the abnormal group and .518 for the normal group. That means that the abnormal group shows 50 per cent of its members are below the grade of 41 per cent of their class, while for the normal group 50 per cent are above 51.8 per cent of their class. This would seem to indicate an important difference in ability correlated with the two groups. If we had the hyperthyroid cases separated from the hypothyroid cases the

[18] Psych. PR. Percentile Rank on American Council on Education Psychological Examination, based on the scores of all freshmen entering the University of Oregon in that year. It indicates the percentage of the freshmen class who rank below the individual whose PR is considered: .00 PR is at the bottom of the class, .50 PR at the median, .99 at the top.

difference in the medians of the two groups would be still more startling. If a group is characterized by retardation in physical characteristics it is quite possibly retarded in mental too.

When the point-hour score,[14] GPR, is used as a basis of comparison we find that the average for the abnormal group is 2.91, while that for the normal is 3.02. Since these scores range from the lowest at 1 to the highest at 6 we find the abnormal group still low. The P. E. of the difference of the means is .06 while the difference of $M_1M_2 = .11$. These Psych PR medians are the more striking when one compares the men with a median of 49.5 for the men of this class with the median for the women of 56.2. This higher median for the women is achieved when just over 21 per cent of them have a median of .41. The women students would seem on the whole to be a group quite superior to the men especially in view of their handicap due to thyroid disorder.

A further index of difference between the two groups is the academic mortality, or withdrawal from college for all reasons. The withdrawals are taken at the end of the fall quarter, 1930-31, the time when this study was under way. Economic considerations should have some influence here, but unless striking differences were shown in the occupational status of the two groups we should expect the economic factor to be constant. There are but two cases of any differences at all worth noting in occupational status, manufacturing and mechanical industries and professional service, yet the elimination from college as shown by the two groups shows no general similarity to the occupational differences for these two categories.[15]

The total academic mortality after four quarters is shown to be 43.7 per cent for these women. The normal thyroid group, which composed 79 per cent of our total group, provided 73.2 per cent of the withdrawals and the abnormal group 26.8 per cent of the withdrawals, although it composed but 21 per cent of the original number.

When we compare these two groups on the basis of the per cent of each group eliminated, the results are still more striking.[16] Of the total number showing normal thyroids we find 40.5 per cent are no longer in the University after four quarters, but of the abnormal group 55.7 per cent have withdrawn. There is thus seen to be a much heavier elimination in the abnormal thyroid than in the normal thyroid group. This difference is probably due to conditions incident to the thyroid

[14] Points are given as follows: 5 points for each term-hour of grade I, 4 for II, etc., to 0 for F or conditioned. These are summed for all courses in which

disorder. Academically the abnormal thyroid group shows a status inferior to the normal group.

In conclusion we can say that thyroid disorder as reflected in our study of 375 cases shows certain results corroborative of clinical suggestions. The evidence of faulty metabolism, greater inconvenience during menstruation, and lower academic achievement is all cumulative. The results are not final but are surely indicative of points deserving further study.

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the student received one of these grades. GPR is a quality index with a possible range from 0.00 as the minimum to 5.00 as the maximum.

[15] *Distribution by Occupations of Father and Academic Mortality by Occupation*

Occupations of Father	*Occupational Distribution*				*Academic Mortality*			
	Normal		Abnormal		Normal		Abnormal	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
I. Agriculture Forestry and Animal Husbandry	54	18.3	13	16.5	24	20.1	7	15.9
II. Extraction of Minerals	0	0.0	0	0.0	0	0.0	0	0.0
III. Manufacturing and Mechanical Industries	23	7.7	9	11.4	7	5.8	6	13.6
IV. Transportation	18	6.0	3	3.8	9	7.5	1	2.3
V. Trade	80	27.0	21	26.6	38	31.7	9	20.4
VI. Public Service	10	3.4	2	2.5	1	0.8	1	2.3
VII. Professional Service	47	15.9	10	12.6	9	7.5	9	20.4
VIII. Domestic and Personal Service	7	2.4	3	3.8	3	2.5	1	2.3
IX. Clerical Occupations	2	0.7	0	0.0	1	0.8	0	0.0
X. Miscellaneous	55	18.6	18	22.8	28	23.3	10	22.8
Total	296	100.0	79	100.0	120	100.0	44	100.0

[16] *Academic Mortality by Type of Thyroid*

	Registered in University 1929-30		*Withdrawn from University end of Fall Term 1930-31*	
	No	Per cent	No	Per cent
Total	375	100.0	164	43.7
Normal Thyroid	296	100.0	120	40.5
Abnormal Thyroid	79	100.0	44	55.7

STUDIES IN HUMAN INHERITANCE VI

A GENETIC REFUTATION OF THE PRINCIPLES OF "BEHAVIORISTIC" PSYCHOLOGY*

BY DAVID C. RIFE AND LAURENCE H. SNYDER
Department of Zoölogy, Ohio State University

INTRODUCTION

In the progress of any science there are always developed in the way of by-products a certain amount of illogical reasoning, a few radical statements, and some unsound principles. Our own science of genetics has not been free from these, but the heart-wood is sound, and we are ourselves rapidly pruning the dead branches.

It has happened in the science of psychology that a few workers, otherwise apparently clear-headed, have started off on a tangent from the fundamentally sound conception of conditioned reflexes, and by means of further "conditioning," have staked the entire mental development of a child on environment. Such concepts Watson has included in his definition of the term "behaviorism." The term in this sense has little standing among psychologists or biologists, yet it has a broader and more valuable meaning to the field of psychology, and should not be appropriated for any one radical cult. Thus behavioristic psychology and "behavioristic" psychology are quite different. Many psychologists are now behaviorists, though only Watson and his followers are "behaviorists."

It is the contention of the latter group that any normal healthy child can be moulded to any desired pattern: artist or musician, recluse or social celebrity, craven or hero, fool or savant. No very pertinent data have been presented for such a sweeping hypothesis. Such data as are available are largely for the emotional side of development: likes and dislikes, fears and desires. Such characteristics, it may be mentioned in passing, seem to be less strictly circumscribed by genetic make-up than other mental qualities. It is unscientific, however, to extend by hypothesis this "behavioristic" conception to other mental characters: to musical ability, for example, or to genius, or to mathematical or artistic ability.

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* Contributions from the Department of Zoölogy and Entomology, number 108.

But if the evidence for "behaviorism" is scanty, so, it must be admitted, is the evidence against it. Adequate human data in any field of psychology or genetics are at a premium. Geneticists react automatically against "behavioristic" concepts, but have contented themselves largely with theoretic rebuttals. The investigations on identical twins, the correlations of genius in families, and the development and use of I. Q. and other measurements are indirect evidence against these tenets. There is need, however, for a more concise demonstration of the importance of the genetic make-up in the development of mental qualities.

With these thoughts in mind the present investigation was undertaken. It seemed to the authors that direct evidence might be obtained from a genetic study of those individuals who, though feeble-minded, possess to a marked degree some special ability. Such individuals are known to occur, and are generally spoken of as "idiots savants," or "idio-savants." These terms are misleading, as they infer the mental state of idiocy, whereas actually the subjects cover the entire range of feeble-mindedness.

It is known that feeble-mindedness is in most instances hereditary, and that when it is inherited it is due to the action of a single pair of recessive genes. The work of Goddard and others, even when stripped of all doubtful cases, clearly indicates the unit factor basis of this defect. Multiple modifying factors may extend the degree of feeble-mindedness from the idiot through the imbecile to the moron. The higher grade morons merge so smoothly into normalcy as to make classification extremely difficult, especially in the region involving an I. Q. of 70 — 80. The heredity of feeble-mindedness remains, however, clearly based on a pair of unit factors.

Similarly we have evidence that special abilities are hereditary. The work of Mjoen and of Seashore in music, for example, clearly shows the importance of genetic make-up in the development of this trait.

It would seem, therefore, that in the case of the feeble-minded individuals showing marked special abilities, they had received purely coincidentally two sets of hereditary factors, those for feeble-mindedness and those for the special ability. Moreover it would seem that if this is true, the special ability made its appearance because of the genetic make-up of the individual in regard to this trait, and in spite of the absence of normal intelligence. We have seen cases of feeble-minded individuals who were albinos. Both traits showed up at the same time, as would be expected. In similar manner the special ability makes itself

known even in the presence of feeble-mindedness and entirely without training along this line.

The possibility presents itself of obtaining genetic data on this point, thus strengthening the genetic basis for the appearance of special abilities and at the same time bringing definite refutation to bear on the principles of "behavioristic" psychology. Can we find by examining the families of idio-savants that the feeble-mindedness and the special ability both appear, in such a way as to suggest heredity and uphold our point? Can we find feeble-minded relatives of these individuals who do not possess the special ability, and normal relatives who do? It would seem reasonable to expect this from a genetic standpoint.

Let us first review briefly the cases of idio-savants previously recorded. Tredgold, in his book, "Mental Deficiency," devotes a chapter to such individuals. His descriptions show quite a variation in regard to the particular type of ability possessed. For example, some possessed an almost incredible capacity for mechanical work requiring great cunning and dexterity, as in carving and woodwork, and, closely akin to this, many had unusual talent for drawing. There is cited the case of a cretin imbecile, named Gottfried Mind, who was born in Berne, Switzerland, in 1768. At an early age he showed a talent for drawing, and as it was obvious that he would never be able to earn his living in any ordinary occupation, his father's employer provided some training for young Gottfried. He never learned to read and write, he had no idea of the value of money, his hands were unusually large and rough, and his general appearance so showed mental defect that his walks through the city were usually to the accompaniment of a crowd of jeering children. In spite of all this, his drawings and water color sketches of various animals were so lifelike and well done that he acquired a European fame.

Other cases are mentioned where the special ability was some unusual type of memory. One of these, a high grade imbecile, could, if given any date during the last five years, state the day of the week correctly without any hesitation. He could also give the product of any two numbers under twenty with "the rapidity of a reflex movement." Another case of the same type was that of an inmate of an idiot asylum in Vienna who could immediately tell what day of the week it had been, or would be, on any given date of the month in any year during the long period from the year 1000 A. D., until the year 2000 A. D. For example, if asked "What day of the week was October 3, 1907?" he

would immediately say Thursday, which is the correct answer. There is recorded the case of an imbecile who could repeat verbatim a newspaper he had just read, and a more remarkable patient who could repeat backwards what he had just read.

Some individuals possessing unusual musical ability are also mentioned. There are idiots, unable to speak a single word, who could hum a tune which they had heard only once, with perfect accuracy. A more remarkable case was that of "an imbecile born blind, afflicted with rickets, and crippled, who had great musical talents. Her voice was very correct, and whenever she had sung or heard some piece of music, she knew perfectly well the words and the music. As long as she lived they came to her to correct the mistakes in singing of her companions, or asked her to repeat a passage which had gone wrong, which she did admirably."

The most interesting cases recorded are of feeble-minded individuals with extraordinary capacity for arithmetic and calculations. There was an imbecile boy, twelve years of age, who could multiply three figures by three figures with lightning rapidity, and another low grade patient, who, if told the age of anyone, could in a very short time calculate the number of minutes he had lived. There is also mentioned an imbecile girl who had a remarkable faculty for multiplication and division. For instance, "she divided mentally 1576, 560 and 336 by 16 with astonishing quickness; she multiplied such numbers as 23×23; 45×18; 78×78 almost immediately and by a peculiar method of her own. And yet in addition and subtraction she was remarkably poor, and said that 57 added to 63 gave 141, 48 and 153 gave 163, etc." Most interesting of all was the case of a young man completely blind, who could give the square root of any number running into four figures in an average of four seconds, and the cube root of any number running into six figures in six seconds. When he was asked how many grains of corn there would be in any one of sixty-four boxes, with one in the first, two in the second, four in the third, eight in the fourth and so on, he gave the answers for the fourteenth (8,192), for the eighteenth (131,072), and the twenty-fourth (8,388,608) instantaneously, and he gave the figures for the forty-eighth box (140,737,488,355,328) in six seconds. He also gave the total in all sixty-four boxes correctly in forty-five seconds.

Probably the best known case was that of an inmate of the Earlswood Asylum in England, who was known as the "Genius of Earlswood

Asylum." This case has been described in some detail by Tredgold. The Genius of Earlswood Asylum had a marvelous skill in drawing and mechanical dexterity, probably unequalled by any other case. He was born in 1853, and no other record of feeble-mindedness has been found in his family. His parents, however, were first cousins. He was always very deaf, and it is interesting to note that he had one brother of normal mentality, who was deaf and dumb and possessed a greater aptitude for drawing than the patient. As no school would take him, his only education was received at home, and practically all he learned was to write and spell the names of simple objects. He was admitted to Earlswood Asylum at fifteen years of age, and at that time was described as having a good memory and power of imitation, and as being fond of drawing and examining how things were made. As a result he was put to work in the carpenter's shop and "soon became an expert craftsman." During the remainder of his life, spent in the Asylum, he completed a group of crayon drawings, carvings in ivory and wood, models of ships, etc., which today adorn the walls and fill two large workrooms. The most wonderful of these is the model of a steamship which he named the Great Eastern. Tredgold gives the following account of this work:

> It took him three years and three months to complete every detail, including brass anchors, screw pulley-blocks and copper paddles, all of which were actually made by the patient himself, from drawings he had prepared beforehand. The planks of this leviathan are fixed to the ribs by wooden pins to the number of nearly a million and a quarter. All of these were made by Pullen (the patient) in a special instrument, which in turn he also planned and made. He also devised and executed a strong carriage on four wheels for the conveyance of the ship. The model is ten feet long, eighteen and five-eighths inches wide, and thirteen and five-eighths inches in depth. It contains 5,585 copper rivets, and there are thirteen lifeboats hoisted on complete davits, each of which is a perfectly finished model. It is fitted with paddles, screws and engines, and it contains staterooms, which are decorated and furnished with chairs, tables, beds and trunks. He also invented and attached an arrangement of pulleys by which the whole upper deck may be raised so as to show the parts below. I believe that when first put in the water, the huge model capsized, but that has since been remedied.

In conclusion Tredgold says: "His powers of observation, comparison, attention, memory, will and pertinacity, were extraordinary, as is shown by the foregoing account; and yet he was obviously too childish and at the same time too emotional, unstable and lacking in mental balance, to make any headway, or even to hold his own in the outside world. Without someone to stage manage him, his remarkable gifts would never have sufficed to supply him with the necessities of life."

All of the cases described by Tredgold were found in the British Isles and Europe, but cases of the same type have existed and still exist in the United States. Dr. Goddard tells us of a case he knew in the Institution in Vineland, N. J., several years ago; that of a blind imbecile girl who possessed unusual musical ability. She could play a new, difficult piece on the piano after having heard it only once. On one occasion a rather noted musician was visiting the Institution, and when told about the girl, requested that he might see for himself what she could do. When she appeared he played an unpublished composition of his own, and then asked her if she could play it. She asked to hear it once more, and after it had been played the second time, she played the piece perfectly. Another case of the same type was that of "Blind Tom," a blind negro of the imbecile grade, who was an unusual pianist. He used to make many public appearances, and after playing a piece would applaud himself. "Blind Joe," another musician of the same type, used to appear in Keith's vaudeville.

There are also quite a number of feeble-minded individuals possessing memory for certain events, dates, etc. In an Ohio town near us there lives a feeble-minded boy, probably of imbecile mentality, who has memorized the telephone directory, and also has a passion for learning the numbers on automobile license tags. He often performs before audiences, giving the telephone number or license tag number of local persons, when asked to do so. There lived in Indiana several years ago, a boy who had an amazing capacity for remembering numbers. No data have been found in regard to his exact mental age, but judging from the records, he was probably a high grade moron. He would go to a railway crossing, and if a freight train passed by, would be able to repeat from memory the numbers on all the cars, after it had passed. As these numbers often run up into the tens and hundreds of thousands, this was an astonishing feat, to say the least. Bryan and Lindley, of Indiana University, studied this case, but before they had arrived at any definite conclusions, his father took him into vaudeville, to exhibit his ability.

EXPERIMENTAL RESULTS

The first step in attacking this problem was the sending of letters to the superintendents of the various institutions for the feeble-minded in the United States, explaining the nature of the investigation, and enclosing an inquiry blank. This contained spaces for the recording of

any inmates with special abilities, with information as to mental age, chronological age, special ability, and family history.

Fifty-five such letters were sent to as many institutions, and forty replies were received. Twenty-four of these were negative, while the remaining sixteen sent records of a total of thirty-three cases. Of these, eight were of the musical type, eight were mathematically inclined, seven possessed unusual ability for drawing, and the rest were of various types, including mechanical ability, phenomenal memory, and so on. In some of these no knowledge could be obtained of the family history.

It was hoped to see personally all of the cases which appeared to be of interest, and examine the families. It proved feasible, however, to see personally only those in the Institution at Letchworth Village, New York. The other cases were continued by careful correspondence with the physician in charge. Nine cases were personally interviewed at Letchworth Village, and their families examined at their own homes where feasible. These cases will be considered first.

An interesting example is that of a negro boy, eighteen years old, but a low grade moron. The mother is immoral and unstable, the father a brutal type of border-line mentality. This boy has a special ability for drawing, and readily drew excellent pictures of such objects as he could understand. He has a brother in the same institution, feeble-minded, but with no drawing ability at all. Another normal brother who attends high school has the exceptional drawing ability.

Figure 1 gives this family history in chart form.

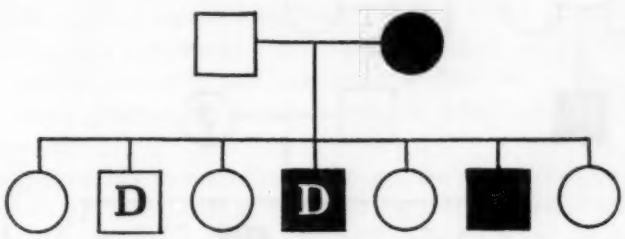

FIG. 1. D INDICATES DRAWING ABILITY. SOLID SQUARES AND CIRCLES INDICATE FEEBLEMINDEDNESS

A second case is that of a twenty-seven year old Jew with a mental age of three years. He has a special mathematical ability, which appeared very early. As a small child he would scribble figures on the bath-

room tiles or other places whenever he could get hold of a pencil. He never learned to talk, and even now cannot perform such simple requests as pointing to his eyes or ears. In school he could do absolutely nothing, so was sent home, and at sixteen was admitted to the Institution. His hearing is normal.

Although he is incapable of carrying on a conversation, or of understanding spoken requests, one may make one's desires along mathematical lines known with a pencil. When a pencil and paper were taken, and the figures 2, 4 and 8 written in a vertical column, the patient immediately continued the series 16, 32, 64, etc. When the series 2, 4, 16 was started, he immediately continued this one, the sixth number being 4,294,967,296. Then $9-3$ was written, in the attempt to indicate square root. Under this several numbers such as 625, 729, and 900 were written. The square root of each was immediately and correctly written. Any problem in multiplication of several digits by several digits was done immediately, only the answer being written. In problems of addition, however, the patient was very poor, giving incorrect answers to simple problems.

This boy's father has a feeble-minded first cousin. No outstanding mathematical ability is found among the family, although the mother is fond of and good at mental arithmetic.

Figure 2 shows the condition in this family.

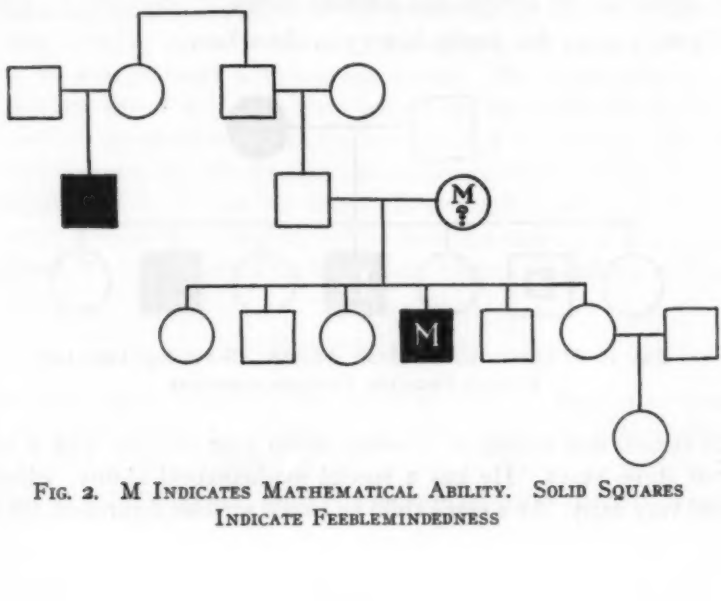

FIG. 2. M INDICATES MATHEMATICAL ABILITY. SOLID SQUARES INDICATE FEEBLEMINDEDNESS

Case 3 is an example involving inherited musical ability but this time expressed in the presence of acquired feeble-mindedness.* The patient is thirty years old, but a low grade moron. The feeble-mindedness is said to be due to spinal meningitis, which occurred at three years of age. The patient has a sister who is a gifted violinist. His paternal grandmother had unusual musical ability as a pianist. Two first cousins show exceptional musical ability. The rest of the family do not exhibit any special talent along this line, although training was given to some of them.

The patient himself, in spite of his lack of mental ability, plays either classical music or jazz, with many variations. His ability is plainly inherent, and not the result of training.

The family history of this case is given in Figure 3.

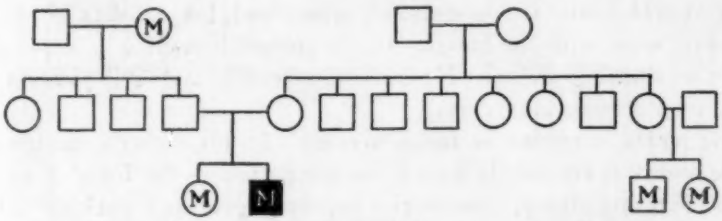

FIG. 3. M INDICATES MUSICAL ABILITY. SOLID SQUARE INDICATES FEEBLEMINDEDNESS

Case 4 is a boy of twenty-one, but a low grade moron. He has marked mechanical ability, and loves to tinker with any kind of machinery. When given a mixed assortment of door locks, bicycle bells, and other mechanical contrivances with which he had not worked before, he had them all correctly put together in a short time. He was given the Stenquest Mechanical Aptitude test, in which he was rated as a superior adult.

The patient has two brothers who are feeble-minded, but show no ability at all along mechanical lines. A sister is also feeble-minded with no mechanical ability. Another sister is too young to be determined. The father and mother are both feeble-minded, but in spite of this the father showed such aptitude with tools that under supervision he earned a good salary as a plumber's helper. This indicates something of the same mechanical ability.

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* This case was written up by Minogue, *J. Appl. Psychol.* 7: 349, 1923.

Figure 4 shows this family in detail.

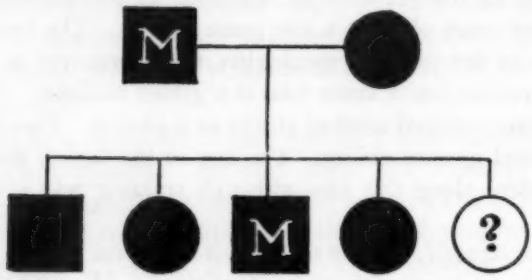

FIG. 4. M INDICATES MECHANICAL ABILITY. SOLID SQUARES AND CIRCLES INDICATE FEEBLEMINDEDNESS

Case 5 is that of a white man, thirty years old, but an imbecile. The ability of this family is less definitely specialized, but consists of ability to do fine work with the hands. In the patient himself it is expressed largely as drawing ability. He can draw readily and well pictures of well-known persons and objects.

The patient's mother is feeble-minded. In his father's family the artistic ability is frequently found, sometimes taking the form of painting and lettering ability, fine interior finishing, gold leaf working, lithographic ability, and so on.

This family history is shown in Figure 5.

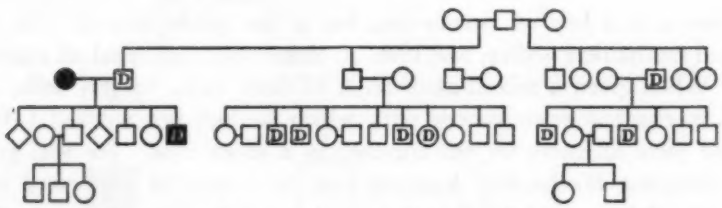

FIG. 5. D INDICATES ARTISTIC ABILITY, USUALLY TAKING THE FORM OF DRAWING ABILITY, BUT SOMETIMES OF OTHER FINE MANUAL ARTISANSHIP. SOLID SQUARES AND CIRCLES INDICATE FEEBLEMINDEDNESS

Several cases were seen at the Institution, in which a phenomenal memory manifested itself in the presence of feeble-mindedness. Although nothing could be learned of the family histories in these cases, it is clear that no training was involved in the development of this ability.

An interesting case was that of a low grade idiot, unable to speak a word or dress himself. Yet when given an ordinary corrugated dust-pan, he spun it rapidly, balancing it on the index finger. Other objects could be similarly spun, using either hand. His parents were both vaudeville actors.

Of the cases followed up by correspondence with the attending physicians, several gave points of interest. One case is of a woman of thirty-five, but an idiot, in the Michigan Home and Training School. She plays the piano by ear, being able to play any tune sung to her. Her chords are correct from the standpoint of harmony. She has had no

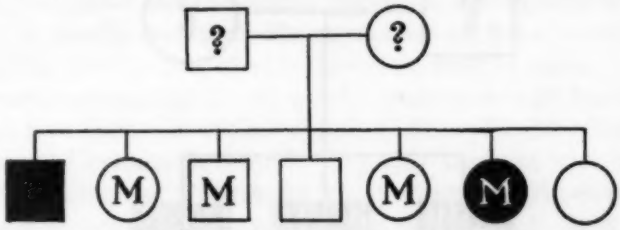

FIG. 6. M INDICATES MUSICAL ABILITY. SOLID SQUARES AND CIRCLES INDICATE FEEBLEMINDEDNESS

training in music. She has an idiot brother. Some of her normal brothers and sisters play and sing, and one sister does vocal work in the community. Here both the special ability and the feeble-mindedness are found in the patient's relatives.

This case is illustrated in Figure 6.

Another case at the Wrentham, Massachusetts, State School is an idiot boy of nineteen years, blind, who plays the piano by ear. He plays

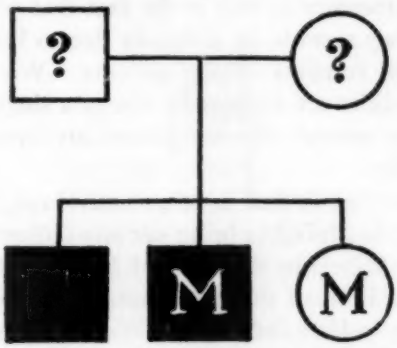

FIG. 7. M INDICATES MUSICAL ABILITY. SOLID SQUARES INDICATE FEEBLEMINDEDNESS

anything he hears, and is said to have perfect pitch. He has a feeble-minded brother who has no musical ability, and a normal sister, who, though blind, plays the piano and composes music.

This interesting family is illustrated in Figure 7.

There is also a man at this Institution with a mental age of two years, making him an idiot. He exhibits a special talent for drawing. He has two feeble-minded brothers with no drawing ability, in the same institution. His family is illustrated in Figure 8.

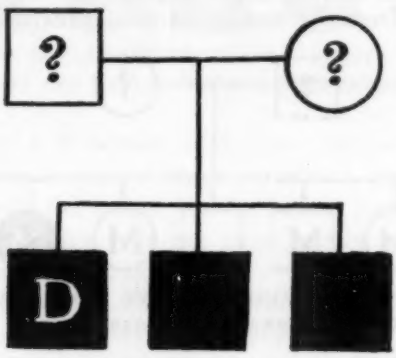

FIG. 8. D INDICATES DRAWING ABILITY. SOLID SQUARES INDICATE FEEBLEMINDEDNESS

CONCLUSIONS

Several things are clearly evident from this study. One is that special abilities may develop to a marked extent in the presence of feeble-mindedness, even idiocy, and quite in the absence of training or instruction. Supplementary to this is the fact that where such special abilities do occur, they seem to be definitely due to heredity, since they appear frequently in relatives of the patients. We do not mean to infer that special abilities are necessarily due to a simple type of inheritance. Undoubtedly several separate factors are involved in the production of each ability.

Another apparent fact is that in other individuals, both normal and sub-normal, training has failed to bring out any indication of the special ability. This is also upheld by the work of Mjoen and others.

It must be borne in mind that these special abilities are not in any way connected with feeble-mindedness. We all know normal persons who have marked ability along musical, mathematical, artistic or mechan-

ical lines. Thus all combinations of normal mentality, feeble-mindedness, special abilities and their lack may occur. Special abilities are inherited quite independently of general intelligence. It is merely a coincidence when a person inherits both. The fact that even feeble-mindedness does not nullify the appearance of a special ability in those who inherit it, and that training is not essential for its appearance (though it would be, of course, for its complete development), coupled with the fact that even training fails to bring out such ability in other individuals, would seem a specific refutation of the principles of "behavioristic" psychology.

One final point may be made. Although the importance of genic make-up is clearly evident in the appearance of these special abilities, the complete development of such characters must of course be dependent on environment, that is, on specific opportunity and training along these various lines. A radical stand at either the hereditary or the environmental extreme is absurd. We may think of such characters, in the terminology of Terman, as $x - y$ characters, dependent on the interaction of two variables, heredity and environment. It is the interesting task of the geneticist and psychologist, working together, to solve the various personal equations for these unknowns.

We wish to take this opportunity to thank the many officials of institutions who so kindly replied to our letters, and especially to the officials at Letchworth Village who so heartily coöperated with us in the interviews with the families of patients.

CHANGES IN THE SEX RATIO IN INFANT MORTALITY ACCORDING TO AGE

BY S. J. HOLMES AND V. P. MENTZER

In a paper on "The Sex Ratio in Infant Mortality as an Index of a Selective Death Rate" (1926) it was pointed out by one of the writers that, while there is a reduction of the sex ratio in infant deaths (the proportion of males to 100 females) as we pass from birth to the end of the first year, there is an actual increase in this ratio for a short time after the first month. Quite independently the same fact was mentioned by Dr. T. H. C. Stevenson for infant deaths in England and Wales for the period 1905-1922. The data cited by Dr. Stevenson are as follows:

Sex Ratio at Death (Males to 100 Females) of Infants According to Age by Months in England and Wales, 1905-1922

Month	1	2	3	4	5	6	7	8	9	10	11	12	Total
Ratio	130	134	127	123	121	125	122	120	116	115	113	109	125

In his discussion of the course of infant mortality in England and Wales, Dr. Stevenson remarked, "From these average figures it will be seen that the excess mortality of males is almost fully manifest from the moment of birth, increasing very slightly to a maximum about the end of the first or beginning of the second month of life, after which it rapidly and on the whole steadily, declines to comparatively small dimensions at the end of the first year."

Although the rise of the sex ratio after the first month was exemplified by a large number of cases, from the United States as well as England and Wales, it was thought desirable to subject the matter to a further test by making a comparison of the data on infant mortality according to month in various countries. The results appeared at first quite contradictory and seemed to throw doubt upon the generalization made upon the basis of British and American statistics. From an inspection of Table 1 it will be apparent that while in some cases the sex ratio at death is higher in the second month than it is in the first, in several others it is considerably lower. Inasmuch as the ratios for several of the countries considered are based on several thousand or even

several hundred thousand cases, the differences cannot be due to mere chance. Either there must be some source of error affecting the compilation of statistics of the various countries in different ways, or else there must be some real biological basis for the discrepancies.* The latter conclusion, we have come to believe, is the correct one.

In dealing with the available material, the first step is, naturally, the tabulation and summation of male and female deaths in the various countries for subdivisions of the first year. Not all countries subdivide the first year in the same way. The U. S. Birth Statistics from 1917 to 1921 give deaths by months for the first three months, and by quarters for the rest of the year, but in 1922 and thereafter deaths are given for each month of age. Deaths are also given for subdivisions of the first month, and even by days for the first few days of the first week. A few other countries now publish deaths for each month of the year, as well as for each of the first few days of the first month. Several countries record deaths under one month of age, one to three months, and either for three to twelve months, or for the second quarter and last half of the year. Since the sex ratio after the third month shows in general a downward course, we have combined in the table the statistics for the last three quarters of the year.

Inasmuch as more boys than girls are born, and since the two sexes die at different rates in different months, it is necessary to apply correction factors to the raw data in order to ascertain more precisely the true relative mortality of the two sexes for subdivisions of the first year. Knowing the number of boys and girls born, and the number dying in the first month, one can estimate the death rate of each sex per thousand of that sex born. The true relative mortality of the sexes can then be ascertained by dividing the male death rate by the female death rate. For estimating the mortality of the next period one can start with the survivors of each sex remaining after the deaths in the first month are deducted, and calculate the relative mortality of the sexes in the second period with these survivors as a base. The rates for subsequent periods may be calculated by a similar method. The ratios

* One of the sources of discrepancy arising out of the compilation of statistics is the varying customs of reporting stillbirths. Holland and Belgium, for instance, return as stillbirths all unregistered infants who died before three days of age. Most other countries count as stillbirths only those infants who fail to manifest signs of life after delivery. In general, the ratios here given would be very little affected by the source of discrepancy we have mentioned.

TABLE 1

Infant Mortality Rates, Sex Ratios at Birth, and Sex Ratios at Death for Subdivisions of the First Year of Life

COUNTRY	1	2	3	4			5		
	SEX RATIO AT BIRTH	INFANT MORTALITY PER 1000 BIRTHS	SEX RATIO AT DEATH UNDER 1 YEAR	CRUDE SEX RATIO AT DEATH—MONTH			TRUE SEX RATIO AT DEATH—MONTH		
				-1	2-3	4-12	-1	2-3	4-12
Australia—1917-27	105.6	59.1	132.9	136.3	137.3	126.3	129.1	131.1	120.9
Austria—1914-21	105.8	176.0	125.4	133.5	128.0	116.5	126.5	123.0	112.9
Belgium—1919-25	105.3	100.7	132.5	135.8	137.2	128.3	129.0	131.3	123.6
Denmark—1916-26	105.8	86.2	135.1	138.7	137.3	131.1	131.6	131.4	126.1
Egypt—1919-28	108.3	143.9	133.7	139.6	133.3	132.6	128.8	123.7	123.6
England & Wales—1916-29	104.8	81.9	135.0	136.2	141.9	130.7	129.9	136.7	126.6
Finland—1916-25	106.3	104.6	126.1	129.7	128.9	122.3	122.05	122.18	116.5
France—1914-25	105.1	106.3	128.5	133.1	130.9	123.7	126.9	125.7	119.5
Germany—1917-25	107.1	128.6	130.0	133.6	133.4	125.2	124.8	126.0	119.1
Hungary—1916-26	106.8	180.6	124.0	128.9	123.4	119.3	120.5	117.3	114.2
Holland—1917-25	105.7	71.5	134.1	141.6	141.2	127.4	133.7	134.1	121.5
Japan—1918-25	104.3	164.1	115.3	117.7	119.5	112.4	112.9	115.6	109.4
New Zealand—1917-28	105.7	47.4	134.4	137.2	140.1	123.9	129.8	133.7	123.2
Norway—1911-24, 26	105.8	59.86	129.5	132.6	131.0	127.2	125.4	123.8	121.2
Scotland—1916-26	105.1	94.	133.9	136.4	139.5	129.6	129.9	134.1	125.2
Sweden—1914-22	105.8	67.4	133.2	137.9	139.1	126.0	130.3	132.4	120.3
Switzerland—1917-26	105.0	69.0	132.9	136.0	145.0	122.3	128.6	139.4	116.8
United States—									
White—1917-28	106.0	73.7	133.1	137.4	136.2	125.3	129.6	129.9	119.8
Negro—1917-28	103.3	117.0	124.5	129.4	122.4	119.5	125.3	119.9	118.2
Uruguay—1914-26, 28	107.4	105.8	119.7	124.6	123.0	115.7	116.0	115.1	108.6

obtained in this way are shown in column 5 of Table 1. Naturally, the ratios in this column are lower than those obtained by simply dividing the number of male deaths by the number of female deaths, for the reason that, since more boys than girls are born, more of them may be expected to die. The true mortality ratio in the second month is higher in relation to that of the first month than the crude rates shown in column 4, which were obtained by simply dividing the number of male deaths by the number of female deaths. Where in column 4 the ratio for the first period is higher than that of the second, the difference is reduced in column 5. In the case of Germany and Holland, the crude ratio is higher for the first than for the second period, but the true ratio is higher for the second period than for the first. And in countries in which the crude ratios are higher for the second period, the true ratios are relatively still higher. For deaths after three months, the ratios, both crude and true, show in general a striking decline.

Taken at their face value, the data seem to show that for a time after the first month the sex ratio of deaths rises in some countries, while in others it falls. Is any uniformity discernible in this mass of apparently contradictory results? Some suggestion of regularity is afforded, we believe, if we interpret the data in the light of variations in the general rate of infant mortality. With the exception of Norway and Denmark, whose statistics are based on a relatively small number of cases, all the countries with a low infant mortality rate (below 105) show a higher sex ratio at death in the second than in the first period. With the exception of Japan, the countries with a high infant mortality (over 130) show a lower sex ratio at death in the second period. They also tend to show a still lower sex ratio at death after the third month. This may not be so apparent when we compare merely the number of deaths in the two sexes, but it is brought out more clearly when allowance is made for the sex ratio at birth and the sex mortality in previous months.

In order to secure data from a country in which infant mortality is low, and which at the same time furnishes a large number of cases, we have taken the statistics of England and Wales for the four years in which infant mortality reached its lowest point. The results given in the table are the crude sex ratios, but they show a marked uniformity in their trend during the subdivisions of the year.

For the sake of contrast I have calculated the data during four years from a country, Japan, in which the infant mortality is high.

TABLE 2

Sex Ratios at Death in England and Wales During Years of Low Infant Mortality

YEAR	MONTHS	UNDER 1	1 TO 3	3 TO 6	6 TO 12	DEATHS PER 1000 BIRTHS
1923		139.6	147.0	139.0	129.7	69.4
1926		135.1	147.8	139.2	125.2	71.9
1927		135.9	143.3	137.5	132.0	72.2
1928		136.9	147.8	140.7	126.1	65.1

TABLE 3

Sex Ratio in Infant Mortality in Japan According to Month

YEAR	MONTHS	UNDER 1	1	2	3 TO 6	6 TO 12	DEATHS PER 1000 BIRTHS
1921		117.9	121.7	120.8	115.9	110.6	168.3
1925		119.4	122.6	117.2	117.0	111.2	142.4
1926		120.0	118.3	119.9	116.3	112.2	137.5
1927		119.8	117.0	116.5	118.5	113.4	141.7

In these data from Japan it may be seen that the sex ratio changes little for the first three months, that of the second being sometimes greater and sometimes less than that of the first. There is a decline in the later months of the year, but not to so great an extent as in England and Wales, and the sex ratio is much lower throughout. One finds similar relations in comparing the sex ratios of deaths in whites and negroes in the United States. The negro ratio shows a marked drop in the second period (second and third month), while in the whites there is a slight rise. If we calculate the death ratios for the second and third months separately, we find in the negroes a ratio of 118.7 for the second month, and a slightly higher rate, 121.7, for the third. The fourth to sixth months inclusive show a ratio of 119, and the last six months a ratio of 117.9.

The American whites show a smaller rise in the mortality of the second period than most countries with a low infant mortality, but if we calculate the ratios for the second and third months separately, we find a rise in the second month from 129.6 to 131.2, and a fall in the third month to 128.4. The ratios for three to six months and for six to twelve months are, respectively, 125.0, and 116.7.

In the white population of the United States the mortality of the infants born to foreign mothers is considerably higher than that of the infants of native born mothers. Since 1917 our birth statistics give the mortality by months of infants classified according to the birthplace of the mother. In accordance with the relations found in several countries, we were led to anticipate that the sex ratio at death would be higher for the infants of the native born mothers than for those of the foreign born. Accordingly, we compiled all the data available and the results confirmed our supposition. (See Table 4.) The sex ratio at death

TABLE 4

Sex Ratio at Death of Infants of Native Born and Foreign Born Mothers in the United States Registration Area for Births (1917-1928)

MONTHS		MALES	FEMALES	RATIO
Under 1	NB	325,425	236,543	137.6
	FB	95,430	69,756	136.8
1-2	NB	48,569	34,826	139.5
	FB	17,683	13,314	132.8
2-3	NB	36,394	26,971	134.9
	FB	14,778	11,094	133.2
3-4	NB	17,898	13,557	132.0
(1922-28)	FB	6,513	4,946	131.7

among the infants of the foreign born shows a fall in the second and third month, and it is lower throughout than the sex ratio of the native born. Among the infants of native born mothers the sex ratio rises relatively more in the second month than it does in the white population as a whole. The removal of the effect of the infant mortality of foreign born mothers, which is relatively high, reveals more clearly the tendency of the sex ratio at death to rise for a short time after the first month.

We have also calculated the sex ratio at death from the recent German life tables (1924-1926) which cover a period of low infant mortality. These show a rise in the sex ratio in the second month (128.9) as compared with the first (126.1), a fall in the third (126.5), a slight rise in the fourth (127.8), and a decided drop in the fifth (123.9), and also low rates in the subsequent months. The rise of the sex ratio in the fourth month is unusual.

One cannot, of course, be certain of the statistical accuracy of the reports from all countries. Egypt, for instance, a country which has a very high infant mortality rate and an especially high rate for the last three months of the year, reports the lowest rate of all countries for infants dying under one month of age. Since the rates for other countries with a high infant mortality rate are high also in the first month, this very low rate for Egypt should be regarded with suspicion. Whether in some countries deaths of male infants are more apt to be reported, is uncertain, but this probably would not occur in countries with careful registration, such as England and Germany. A more serious source of error is the inaccuracy in recording the precise month of death. On account of different methods of reporting deaths it might happen that more errors of assignment were made in some countries than in others. Such errors would affect sex ratios at death to a certain extent, but they would affect much more the relative mortality rates of infants in the different months of life. With all the sources of error involved, and in face of the fact that the differences in the sex ratios at death in the early months of the first year are as a rule not large, one is not justified in drawing very positive conclusions in the light of present available evidence. We have made the attempt to secure large samples, but even with all the cases we have compiled, the differences in the death ratios in the first and second months are not as reliable as could be desired for some of the smaller countries. Owing to the large number of deaths occurring in the first months a high degree of reliability—that is, of the purely statistical kind—attaches to the sex ratios at this time.

THE EFFECT OF NON-SELECTIVE DEATHS

In discussing the rise of the sex ratio at death for a short period after the second month in the United States and Great Britain, it is pointed out in the paper referred to (Holmes, 1926) that, while infants become weaker and the male apparently relatively weaker than the female, as we pass back toward the period of birth, there is, nevertheless, in the first month a considerable proportion of deaths from accidental causes. Relatively to later periods there are more deaths from suffocation, homicide, and several fortuitous causes in which the factor of innate vitality plays a relatively small part. We should expect such causes to affect males and females in about the proportion in which the sexes occur at birth. As a matter of fact, the sex ratio of infants from

such causes is low. In 16,783 deaths from suffocation under three months in England and Wales, there was a sex ratio of 106.4, and in 22,248 deaths from violence a sex ratio of 107.7. Deaths from external causes in 17,106 American white infants gave a sex ratio of 123.3, as compared with a ratio of 131 for all causes combined.

If we grant that there is a tendency for the sex ratio at death to rise for a short period after birth, it is possible to interpret the phenomenon in more than one way. It might be assumed, for instance, that, owing to developmental causes, the disparity in the innate vitality of the two sexes actually increases for a time after birth, and that the removal of mainly non-selective causes of death simply permits this disparity to reveal itself more clearly. On the other hand, we may interpret the facts in harmony with the assumption that the relative frailty of the male is greatest at birth and gradually approaches equality with that of the opposite sex with increasing age. In this case one would have to assume that the rise of the sex ratio at death was due to the greater removal of non-selective causes of death in the second than in the first month of infancy. In countries with a low infant mortality the first month would still include a considerable proportion of non-selective deaths which would tend to decrease the sex ratio at this time. Our second alternative has at least the merit of interpreting the phenomena in accordance with a fairly uniform trend in the relative vitality of the sexes. In choosing between these alternatives attention may be called to the fact that in all the variations in infant death rates in different countries the mortality of the first month has been affected the least. It is well known that reduction of infant mortality has taken place mainly in the later months of the first year. In Table 5 we have given the relative variability in the mortality rates of a number of countries at different times. The countries included Australia (1917, 19-27), Belgium (25, 27, 28), Canada (21-23, 25, 27), Egypt (25-28), England and Wales (17-22), Ireland, including Irish Free State (17, 19-21, 25-28), Northern Ireland (25-28), Japan (17, 19-22, 25-27), Holland (19-22, 25, 27, 28), New Zealand (26-28), Scotland (17-21, 23-28), Switzerland (26-28), United States (17-28), and Uruguay (20, 21, 23, 25, 26, 28). The items compared were the death rates in a given country for various subdivisions of the first year per 1,000 of that sex born. These rates were naturally based on different numbers of cases, but as the object was to study the variability of mortality under different conditions, the items were given the same weight. This procedure some-

TABLE 5

Variability of Infant Mortality Rates of the Sexes per 1000 Living Births

	AGE—UNDER 1 MONTH		1-2 MONTHS		2-3 MONTHS		3-6 MONTHS		6-12 MONTHS	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Mean Death Rate per 1000 Births	39.61	31.42	9.49	7.54	7.50	5.99	16.96	14.04	24.04	20.90
Average Deviation	9.03 ± .32	7.76 ± .28	2.82 ± .10	2.49 ± .09	2.28 ± .08	2.06 ± .07	5.64 ± .20	5.09 ± .18	9.59 ± .34	8.73 ± .31
Relative Variability $\frac{\text{Av. Dev.} \times 100}{\text{Mean}}$	22.79	24.71	29.68	33.04	30.47	34.38	33.24	36.23	39.87	41.76

what reduces the significance of the probable errors which were calculated, but these errors were relatively small. Owing to changes in age groupings in the statistics of the several countries, and to certain omissions in sources from which the figures were derived, data were not at hand for calculating rates for all the individual months in all the countries, so that the several countries were represented for different numbers of years. This method of dealing with the material gives only a rough measure of the actual variation of infant mortality rates, but it nevertheless brings out quite clearly the general fact that variations in infant mortality are relatively low in the first month, and increase throughout the year. There is a rather sudden rise in the variability of the second as compared with that of the first month, a relatively slight increase in the third over the second month, but a rather more decided increase in the last six months. These relations confirm the general impression one gets by going over the infant mortality rates of a number of countries. The rates for particular subdivisions of the year would doubtless be found to vary considerably with a different selection of times and countries, but the general trend during the year would probably be much the same. The same general trend is indicated by a consideration of the mortality rates in somewhat different subdivisions of the year shown in Table 1. These figures are based on a relatively larger number of deaths, but the annual variations in infant mortality are submerged in the general totals. It may be noted that for each age group the coefficient of variability is significantly higher for females than for males.

Environment makes greater differences in the death rates of infants as they increase in age. Since death rates fluctuate more widely in the second period than in the first month, one may conclude that improvement of sanitary conditions removes more causes of death in the second period than in the first. It is not improbable, therefore, that this reduction of causes of death eliminates more that are non-selective in their action. If so, it would cause the death rate which remains to be relatively higher among the apparently weaker males. In the neo-natal period there is probably in all countries a good deal of indiscriminate mortality which is as yet little affected by the many factors which have lowered the infant death rate. How greatly neo-natal death rates may be reduced by the proper procedures it would be unwise to predict. If the hopes sometimes cherished are realized, one might find a rise in the sex ratio at death until it came to exceed the ratio for the second

and third month. During recent years the mortality of the second and third month has been more amenable to attack than that of the first, so that we might suppose that where infant mortality is low the inherent differences in the vitality of the two sexes more adequately manifest themselves at this time, although they may be no greater than in the earlier period.

There are many causes which may change the mortality rate between the first and the third months. Changes in feeding, the diminution of passive immunities derived from the mother, and various other factors expose infants within this short period to quite different kinds of risk. The problem is complicated in many ways. It is not improbable that the sex ratio at death may be influenced by a fall of the birth rate. Countries in which the birth rate is low naturally have a relatively large proportion of first-born children. The first birth is more apt to be attended with difficulty than the second or third birth, and for this reason the infant is more apt to suffer injury. Both the stillbirth rate and the infant mortality rate are relatively lower for the second and third births than for first births, but both rates increase after the birth of several children. Deaths which result directly or indirectly from difficulties of parturition are prone to occur with much greater frequency in male infants. Consequently, in countries in which the birth rate is low and in which, therefore, there is a relatively large proportion of first-born children, the sex ratio at death would be apt to be somewhat raised. This tendency is offset to a certain extent, however, by the relatively larger number of children who are late arrivals in large families and who would also suffer from difficulties of delivery.

Another factor influencing the sex ratio at death is the extent to which syphilis is disseminated in the population. Syphilis occupies a peculiar position among infectious diseases, inasmuch as it acts with especial severity upon infants either before or soon after birth. It not only affects the sex ratio at death directly, but also indirectly by lowering the birth rate.

While the sex ratio of early deaths is affected by the two causes we have just mentioned, it is not apparent why either of them should make the ratio higher in the second month than in the first. Difficulties of parturition affect the mortality of the first month much more than that of the second. Infant deaths ascribed to injuries at birth are about one hundred times as common under one month of age as in the second month. These difficulties are among the causes which tend to keep the

sex ratio of neo-natal deaths exceptionally high. Perhaps the purely biological influence of a low birth rate, by increasing the relative number of first births, would raise the sex ratio of death more in the first month than in the second. But a low birth rate is associated with many other factors whose influence complicates our problem. It commonly goes along with a low infant mortality, greater age of the mother, and better care of mother and child. It is probably because of unfavorable environment that the death rate of infants from injuries at birth in the United States is considerably higher for infants of foreign born mothers than for those of native born mothers, although it runs consistently much lower for infants of negroes. But leaving the negroes aside as belonging to a different species or sub-species of the genus Homo, it is evident that the factor of adequacy of care which is apt to be more influential in low-birth-rate countries, tends to offset, and probably more than offsets, the influence of the greater proportion of first-born infants. Birth injuries apparently tend to make the sex ratio at death higher in the first month of life than in the second. They would, therefore, tend to obscure the action of the causes, whatever they are, which are responsible for the curious rise in the sex ratio at death in the second month of infancy.

In considering the causes that affect the sex ratios of early deaths one should not lose sight of the influence of racial heredity. If hereditary factors are in part responsible for the different ratios found in the several countries inhabited by the white race, it is impossible to separate their effects from that of the many environmental factors which are certainly influential. Among the infants of American negroes the sex ratio at death is lower than it is in some foreign countries which have a higher rate of infant mortality. This may be an effect of race, since childbearing is relatively easy in negro mothers. It is a striking fact that although both the negroes and our foreign born population suffer from environmental handicaps which cause a high infant mortality, the proportion of deaths caused by injuries at birth are unusually high among the infants of foreign born mothers, while they are relatively low among the infants of negro mothers. In the first month the death rate is much higher and the sex ratio at death is much lower in the negroes than in the whites, but it is perhaps significant that the death rate goes down relatively less in the second month than it does in the whites. Other things equal, a people among whom childbearing is easy might be expected to have a relatively low sex ratio in neo-natal mortality. Accord-

ing to several accounts, childbearing among the Japanese is attended with relatively little difficulty. There may be some connection between this fact and the somewhat exceptional circumstance that the sex ratio at death among the Japanese, despite a rather high infant mortality, is somewhat higher in the second month than it is in the first, although it is low in both.

THE SEX RATIO OF ABORTIONS

When one follows the death rates of the two sexes into intra-uterine life, it is found that, in general, the earlier the abortion the higher the proportion of male deaths. Here, again, a qualification must be introduced, for, although the sex ratio of abortions falls from about the third or fourth month, it rises again before the period of birth. Our birth statistics have included records of stillbirths according to period of gestation since 1922, and for the year 1918. Disregarding the relatively few cases purporting to give the sex ratios of abortions during and before the second month of pregnancy, we find, since 1922, an enormously high sex ratio of 375 to 100, for the third month, a lower, although still high ratio, 210.62, for the fourth month, a ratio of 145 for the fifth month, a considerably lower ratio for the sixth month, and the lowest ratio of all, 118.4 for the seventh month of pregnancy. There is a slight rise in the eighth month, 121; a ratio of 137.9 in the ninth, but a very high sex ratio for delayed births in the tenth month.

It is probable that the sex of early abortions is often incorrectly stated. The diagnosis of sex in an embryo of the third or fourth month is not an easy matter for one who is not an expert. In a study of 1410 abortions Schultz found that the sex ratio of the early stages, although high, was much lower than is commonly stated. As Schultz remarks, "The inexperienced, misled by the size of the clitoris, as well as by other factors, may erroneously determine the female foetus to be a male." In his whole series the sex ratio in the third month was 121.0, in the fourth, 117.5; in the fifth, 109.6; in the sixth, 87.5; in the seventh, 108.5; in the eighth, 133.3; and in the ninth and tenth, 167.6. The sex ratios of the later months of pregnancy were not markedly different from what has been elsewhere reported, and they are even higher than the average ratios for the Registration Area for Births. Since the determination of sex in the later months presents no great difficulty, one would not expect a wide divergence in different series. Schultz does

not state the number of cases upon which the percentages for the several months were calculated. If the abortions were distributed much as they are in the statistics for the Registration Area for Births, the great majority of them would have occurred in the later months. For the Registration Area only about 2, 3 and 5 per cent of abortions occur in the third, fourth and fifth months, respectively, and only about 8 per cent occur in the sixth month. Unless the collections which Schultz studied contained an unusually large percentage of early abortions, the probable errors of the percentages for the early months would be very large. While Schultz's results throw doubt on the accuracy of other statistics, they leave us uncertain as to how reliable the statistics on the sex ratios of early abortions really are, both in Schultz's material and in the much larger material derived from various official sources. The very low sex ratio reported by Schultz for the sixth month is probably not reliable statistically. In an earlier paper based upon about half the number of cases reported in his later one, Schultz attributes the lowest ratio of abortions at this month mainly to chance.

A fairly large series reported upon by one who has paid special attention to the diagnosis of sex is worth more than a much larger number of cases diagnosed for the most part by physicians with little familiarity with the subject. Schultz's material, like that furnished by the Bureau of the Census, shows a fall of the sex ratio through the third to the fifth month of pregnancy, and then a rise before the period of birth. The lowest point, according to the U. S. statistics, occurs in the seventh month instead of the sixth, but in later months both series agree in showing a rise. So far as the sixth month is concerned, the U. S. statistics are probably the more valuable, not only on account of the greater number of cases included, but because errors of diagnosis would at this time be very considerably reduced.

In the light of the existing evidence we are justified in concluding that notwithstanding the existence of a bias for reporting the sex of early abortions as male, the curve of sex mortality in utero shows a sag whose lowest point is reached in the seventh month of pregnancy. Then there is a rise to the period of birth. Deaths occurring in the ninth and tenth month are to a large extent associated with the factor of growth and the difficulties of delivery. Difficult labor, mal-presentation, prolapse and compression of cord, asphyxia and causes affecting the placenta and foetal membranes constitute the major categories of death in the ninth and tenth months of pregnancy. At this time the larger

size and larger head of the males are doubtless influential in raising the sex ratio of deaths. It is noteworthy that the sex ratio of infants dying mainly in the first and second months of the first year from injuries at birth was 161.6 (1914-23) for whites, and 155.9 for negroes. As the period of birth draws near, conditions become relatively more unfavorable for the males. Also, some of the same factors which enhance the male death rate in utero continue to exercise a sort of hangover effect upon the mortality after birth, because if they do not kill the infant before it is born, they so weaken it as to cause it to die a short time after birth. Head injury is a fertile source of mortality. Dr. Eardley Holland found that in non-macerated stillbirths about 40 per cent showed at autopsy evidence of injury to the brain or cerebral membranes.

It would, we believe, be an error to attribute the high male death rate of the late uterine and early neo-natal periods merely to the larger size and larger or harder head of the males. Manifestly, the high male death rate of early abortions cannot be so explained. Whether as a result of recessive sex-linked factors, or because of mere maleness, the male appears to be inherently the weaker sex. The ordeal of birth, quite apart from factors of size, would doubtless be more fatal to males than to females.

CONCLUSION

The foregoing facts show clearly that the course of male mortality does not run smoothly; it has its ups and downs. What changes in the sex ratio may occur between the period of conception and that of the earliest abortion whose sex can be correctly diagnosed, we do not know. So far as we can follow its course, the curve representing the changes of the sex ratio at death starts from a fairly high point, declines to the seventh month of pregnancy, and rises again to the period of birth. Then, especially if the infant death rate is low, it tends to rise in the second and sometimes third month after birth. But in any case, it shows a marked fall during the last three-quarters of the year. In childhood it declines still more slowly. Its course in adolescent or middle life depends to a considerable extent upon external conditions. The ratio of male to female deaths may fall below unity in the 'teens, and again in some countries during the most active child-bearing period. But in old age the curve finally takes an upward course. So far as internal factors influencing vitality are concerned, the male is probably always at a disadvantage to a greater or less degree. The greater female death rate

which occurs in some populations during the 'teens is probably due to differences in environmental exposure; and the higher female death rate in some places during the child-bearing period is mainly a result, directly or indirectly, of the function of reproduction. The greater general death rate of women in this period is due to the internal hereditary factors which determine sex, and not to any greater constitutional weakness of females at this time. Most causes of death (there are some exceptions) give rise to a greater mortality of males in the ages in which females most frequently die from accidents and disorders associated with childbirth. Male inferiority in what may be termed innate vitality is a very persistent characteristic. Whether the disparity between male and female vitality undergoes a fairly even reduction from an early period of development is difficult to ascertain, because the manifestation of such disparity by changes in the sex ratio at death is affected not only by biological factors, but also by social customs and traditions.

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NOTES

FOLK MEDICINE IN RUSSIA

To the undersigned, as a member of the National Conference on Pharmaceutical Research, has been assigned the task of securing material for a paper on "Folk Medicine in Russia."

For the past several months, I have been peering into books at the New York Public Library and the library of the New York Academy of Medicine for material on this most interesting subject.

It occurs to me that some of your readers, either through actual knowledge or by contact with others, may be in a position to give me additional information and material upon which I could base my paper. If any of your readers might be able to send me newspaper clippings, pictures, books discussing the subject, etc., it would be greatly appreciated.

SAMUEL S. DWORKIN,
151 ST. ANNS AVENUE,
NEW YORK CITY, N. Y.

QUOTATIONS

ON THE ALLEGED LETHAL EFFECTS OF SEXUAL CONTINENCE IN THE HOLY LAND*

There are two schools of thought among physiologists, psychologists and psychiatrists regarding the effects upon the human organism of total sexual abstinence. Thus we find so distinguished a medical man as Franz Alexander[1] saying (p. 116): ". . . . excessive restriction of the sexual life is the most general cause of neuroses. Precisely because among all the instinctive functions sexuality is the one subjected to the greatest restrictions, does it receive its prominent position in the etiology of neuroses. The reproach so frequently leveled against psychoanalysis that it gives sexual motives undue prominence in the explanation of pathological mental conditions, should be leveled by society against its own insincere and over-severe sex morality. The excessive part played by sex in the causation of neurotic illness is merely the result of its excessive repression."

On the other hand the British Social Hygiene Committee evolved a body of doctrine on the subject, which was officially converted into dogma by the British Social Hygiene Council on March 22, 1926,[2] and contained the following statements (p. 513):

"There is no evidence either from physiology or from experience that for the unmarried sexual intercourse is a necessity for the maintenance of physical health.

"There is no evidence either from psychology or from experience that for the unmarried sexual intercourse is a necessity for the maintenance of mental health."

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* From the Department of Biology of the School of Hygiene and Public Health of The Johns Hopkins University.

[1] Alexander, F. The Psychoanalysis of the Total Personality. An Application of Freud's Theory of the Ego to Neuroses. Authorized English Translation by B. Glueck and B. D. Lewin. With a Prefatory Note by A. A. Brill. *New York and Washington* (Nerv. and Ment. Dis. Pub. Co.), 1930. Pp. xx + 176.

[2] Continence in relation to social hygiene. A statement prepared by the British Social Hygiene Committee and adopted by the British Social Hygiene Council at their meeting on March 22, 1926. *Jour. Soc. Hyg.*, Vol. 13, pp. 513-524, 1927.

These two quotations, which are of a sufficient modernity, well enough indicate how the issue is drawn. Innumerable additional quotations on each side might be given, but it is not necessary. The essential point is that there is an issue. Some honest and well-intentioned folk are in favor of greater freedom in copulatory athletics, while others, no less well-intentioned and honest, are of the opinion that more restrictions in this department would be better.[3]

The opinion of even the well-born and educated laity is divided on the point. Katherine Bement Davis[4] found (p. 332) that of 1200 unmarried women, all at least five years out of college, 394 were of the opinion, with varying degrees of emphasis, that sex intercourse was necessary for physical and mental health; 622 that it was not necessary; while 184 were either non-committal on the point, or "didn't know," or refused entirely to answer so immodest a question. Thus it appears that of the 1016 spinsters who had definite ideas about copulation and were willing to tell about them, 61.2 per cent were, in the defined sense, against it, and 38.7 per cent for it. But lest these figures should seem to convey a too unenticing picture of these girls we hasten to say that 84.6 per cent of them went on record (p. 357) that married people are justified in having intercourse for purposes other than reproduction; and, *mirabile dictu*, 24.1 per cent of them expressed the conviction (p. 354) that a husband is sometimes justified "in having sex intercourse with a woman or women other than his wife," while 20.7 per cent believe that a wife is sometimes justified in pursuing amatory dalliance to its ultimate conclusion with a man "or men" not her husband.

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[3] It is an odd bit of human behavior that there has grown up during the last twenty years or so a great deal of propagandist activity *pro* and *contra* relative to the desirability of sexual intercourse. One of the major tenets of the birth control propaganda is that copulation is a good thing *per se* and should have some of its potentially disagreeable consequences removed; on the other hand, the propaganda against venereal diseases (euphemistically called "social hygiene") has as one of its major tenets that the surest way to avoid contracting these dread ills is to abstain totally from sexual intercourse, except that which has been medically and clerically certified—this somewhat bitter pill being hopefully, if not very seductively, sugar-coated with the statement that sexual activity is "unnecessary." The curious part about all this is that, in not a few cases, the same identical distinguished *savants* work alternately, and shamelessly, for *both* of these crusades. It is a complicated world in which we live!

[4] Davis, K. B. Factors in the Sex Life of Twenty-two Hundred Women. *New York* (Harper and Bros.), 1929. Pp. xx + 430.

CHRONICLES AND MEMORIALS OF THE REIGN OF RICHARD I.

VOLUME I.

ITINERARIUM PEREGRINORUM ET GESTA REGIS RICARDI;

AUCTORE, UT VIDETUR,

RICARDO, CANONICO SANCTÆ TRINITATIS LONDONIENSIS.

EDITED,

FROM A MS. IN THE LIBRARY OF CORPUS CHRISTI COLLEGE, CAMBRIDGE,

BY

WILLIAM STUBBS, M.A.,

VICAR OF NAVESTOCK; LIBRARIAN TO THE ARCHBISHOP OF CANTERBURY AND KEEPER OF THE MSS. AT LAMBETH; SOMETIME FELLOW OF TRINITY COLLEGE, OXFORD.

PUBLISHED BY THE AUTHORITY OF THE LORDS COMMISSIONERS OF HER MAJESTY'S TREASURY, UNDER THE DIRECTION OF THE MASTER OF THE ROLLS.

LONDON:
LONGMAN, GREEN, LONGMAN, ROBERTS, AND GREEN.

1864.

FIG. 1. TITLE PAGE (REDUCED) OF STUBBS'S EDITION OF THE ITINERARIUM REGIS RICARDI

All this evidence again shows that the whole question is one upon which opinion is divided.

It may further be noted that this issue is not a new one. Augustus promulgated the *Lex Julia et Papia Poppaea*[5] to discourage celibacy. *Caelibes* could not inherit property. A candidate for office who had several children was preferred to one who had fewer. On the other hand ecclesiastical legislators have promoted celibacy at various times and places,[6] though not universally. The history of this matter is one of great interest, but beside the present purpose, except for one point to be discussed later.

"Democritus Junior"[7] did not fail to comb the literature of the ancients for opinions upon so important a subject as the just and fit proportion of venery in the general scheme of human living. He devotes the latter part of *Partition 2, Section 2, Member 3* to a summary of his results, which lead him to this final conclusion:[8] "The extremes being both bad, the medium is to be kept, which cannot easily be determined. Some are better able to sustain, such as are hot and moist, phlegmatic, as Hippocrates insinuateth, some strong and lusty, well fed like Hercules, Proculus the emperor, lusty Laurence, *prostibulum faeminae Messalina* the empress, that by philters, and such kinds of lascivious meats, use all means to enable themselves; and brag of it in the end, *confodi multas enim, occidi vero paucas per ventrem vidisti,* as that Spanish Celestina merrily said: others impotent, of a cold and dry constitution, cannot sustain those gymnics without great hurt done to their own bodies, of which number (though they are very prone to it) are melancholy men for the most part." Burton found opinion divided on the subject as it is now. Thus Avicenna and Oribasius write of the curative effects of copulation in nervous and mental diseases, including the "falling sickness" [epilepsy]. On the same side, *pro copulatione,* are arranged, with references and quotations, Montaltus, Valescus, Mercatus, Rodericus à

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[5] Smith, W. Dictionary of Greek and Roman Antiquities. 3d American edit. 1871, pp. 556, 557.

[6] The great authority here is H. C. Lea's *An Historical Sketch of Sacerdotal Celibacy in the Christian Church.* Second Edit. *Boston* (Houghton, Mifflin and Co.), 1884. Pp. xx + 682. A third edition appeared in 1907.

[7] The Anatomy of Melancholy . . . . . By "Democritus Junior." [Robert Burton.] *London,* 1621 [1st edit.] —1676 [8th edit.]

[8] Quoted from the Chatto and Windus "new edition" of 1898, p. 313, omitting the footnotes. This was a reprint of the 1651-2 edition, which was the last to be corrected by Burton himself before his death.

Castro, Platerus, Marcellus, Donatus, Alexander Benedictus; and on the other side, Lodovicus Antonius, Ficinus, Marsilius Cognatus, and Halyabbas the Arabian and Jason Pratensis, who "make it the fountain of most diseases."

In all the alleged evidence which has been adduced to support one or the other of the positions as to the physiological desirability of celibacy, we have seen little that is statistical, and virtually none that is historical. It is the purpose of this note to make a small contribution of an historical character to this question.

During the four hundred odd years between the time when Jerusalem fell into the hands of the infidel Chosroes in 614 and July 15, 1099, when Godfrey of Bouillon carried his warriors of the First Crusade inside its walls, the state of the Holy Land had been a reproach and a shame to all Christendom. But the decline and final end of the power of the Roman Empire created troubles enough in Europe to keep everyone busy at home. The forging and welding of the European states took all the blood and iron there was about. None was left over with which to wrest Palestine from the clutches of the Moslem. But by the beginning of the eleventh century matters had quieted down greatly. The German Caesarship was in full strength. There was, for the moment, something like a state of equilibrium between the various powers. Scattered pilgrims came back and reported that the Moslem power was waning, just as Rome's had in its day, and for much the same reasons. The idea got about that if a joint expedition could be managed, the long desired end could be accomplished. And so came the Crusades. To the human biologist there are few more interesting events in the history of mankind than the Crusades, and from many and diverse angles. It is not the purpose of this essay, however, to go into that, but more modestly, merely to quote one chapter from one eye-witness account of one Crusade.

Very few detailed and comprehensive accounts of the Crusades by eye-witnesses have been preserved. The three most interesting of such accounts are (1) Richard of Devizes' Chronicle, concerning the deeds of Richard Coeur de Lion; (2) the *Itinerarium* of the same worthy; and (3) de Joinville's memoirs of St. Louis. The second of these, the *Itinerarium Peregrinorum et Gesta Regis Ricardi,* is in many respects the most comprehensive, detailed, and interesting of any first-hand account of any crusade. It was published at about the time of Richard's

death, which occurred in 1199. Stubbs[9] says (p. lxix): "It is just possible, that a first edition or a portion of one was published before the king's death . . . . . It is, however, evident that the whole work, as we have it now, was not published until after Richard's death." The limiting dates, between which the *Itinerarium* was published, and gained wide repute, are 1200 and 1220.

There are in existence several manuscripts of the whole, or considerable parts of this book. The oldest of these is the Cotton MS. Faustina, A. vii (Stubbs' B); the most famous one (because it led to an erroneous attribution of authorship) is the MS. of the Public Library at Cambridge, Ff. 1.25 (Stubbs' A), commonly known as the Vinsauf MS.; the best one, because most complete, is the MS. 129 of Corpus Christi College, Cambridge (Stubbs' C). This is the MS. used by Stubbs in making his definitive edition. There are several other MSS. of parts of the book which need not be listed here.

The authorship of the *Itinerarium* furnishes an interesting tale of the vicissitudes of historical scholarship. It is much too long to relate in full here, but, in brief, the facts are as follows. At various times, and for diverse reasons three different claimants have been put forward for the honor. These are (a) "Ricardus canonicus Sanctae Trinitatis Londoniensis," commonly referred to now as Richard de Templo, (b) Geoffrey de Vinsauf,[10] and (c) "Guido Adduanensis," who, as Stubbs (*loc. cit.*, p. xlv) shows, was most probably "Guido Catalaunensis," otherwise Guy de Bazoches, chanter of the Church of St. Stephen in Chalons on the Marne. In a masterly way Stubbs demonstrates that the claim for "Guido Adduanensis" deserves no serious consideration whatever, and further that the weight of the scholarly evidence is against Geoffrey Vinsauf. This leaves Richard the Canon as the person who probably wrote the book. Furthermore Stubbs shows that he was probably the same person who was elected Prior of the Holy Trinity in 1222. But neither of these probabilities can be demonstrated with absolute certainty to have been the facts. However, Stubbs, who is the foremost authority, accepts the former practically without reser-

[9] Stubbs, W. Chronicles and Memorials of the Reign of Richard I. Vol. I. (See facsimile of title page *infra* for complete citation of this work).

[10] There has been much discussion of the origin of this cognomen, which was latinized as Vinosalva. The matter is of no importance, but the derivation "from the German words *Wein* and *sauffen*, which would imply tippling" makes up in *esprit* what it lacks in probability.

vation, and in this we are content to follow him until new and better evidence pointing in some other direction may appear, if it ever does.

The title page of the definitive modern edition of the *Itinerarium* is shown as Fig. 1.

I wish to quote here, using the Bohn translation,[11] the thirty-fifth chapter of the sixth book of the *Itinerarium*. The translation was made by "the conjoint labours of a classical scholar and a gentleman well read in Mediaeval history." Their translation antedated by some sixteen years the appearance of Stubbs' profound study of this chronicle, and in consequence the brief discussion of the *Itinerarium* in the preface contains a number of errors. The translation, however, is on the whole spirited and accurate, as may be determined by comparing it with the original, which I have done. The translators used as text the inferior edition of Gale, printed in 1687.

Book VI

Ch. XXXV.—*How the pilgrims, having fulfilled their pilgrimage, set sail for their own country, but suffered many shipwrecks and hardships on the way.*

The people had now completed the pilgrimage to which they had devoted themselves, and preparing their fleet to return home, they spread their sails to the winds, and committed themselves to their ships. The fleet speedily set sail, and the ships were wafted in different directions, according to the variety of the winds. For a long time they were tossed about on the waves, and some of them reached different ports in safety; others were driven about, and in danger of being shipwrecked; others, again, died on their voyage, and found their grave in the depths of the ocean; others also were seized with incurable diseases, and never recovered or returned to their own country. Others, moreover, who endured in safety to the end, through the loss of their fathers, brothers, relations, and friends, who had perished of disease or by the sword, are believed to have endured a severe species of martyrdom, and diverse passions pierced their breasts as with a sword. Each, in his own way, we must admit it, endured a kind of martyrdom; every one, in short, of those, who with simple and devout hearts had exposed themselves for the love of God to this distant pilgrimage. Some, however, with loquacious garrulity, were accustomed afterwards to complain that the pilgrims had done little good in the land of Jerusalem, because they had not freed the city; but they did not know what they were saying, for they were inquiring about things of which they have no personal knowledge or experience. We, however, who have seen, and who know all of it by our own eye-sight, claim to be believed in our accounts of the tribulations and miseries

[11] Chronicles of the Crusades, being contemporary narratives of the Crusades of Richard Coeur de Lion, by Richard of Devizes and Geoffrey de Vinsauf; and of the Crusade of Saint Louis, by Lord John de Joinville. *London* (Henry G. Bohn), 1848.

which those men endured. *Wherefore, we state confidently, in the hearing of those also who were present, that 100,000 Christians perished in that pilgrimage, for the sole reason that, in the hope of divine reward, they had separated themselves from women, deeming it wicked by sacrificing their purity to obtain bodily health; and thus they opposed patience even to the corruption of the flesh, that the purity of their minds might remain unimpaired.*[12] We know also, for certain, that by sickness and famine combined, there died more than 300,000, in the siege of Acre, and afterwards, in the same city. Who, however, can doubt of the salvation of the souls of such noble and excellent men, who daily heard divine service from the lips of their own chaplains? These surely may be supposed to have gone to heaven."

There is a great deal more that is of medical and sanitary interest in the *Itinerarium*.[13] But Ch. XXXV seems to make a particularly important contribution to a mooted question. The real problem, of course, is as to what it really was, medically and biologically considered, which led Richard the Canon to make this statement about the 100,000. For this problem I have no final solution, but to dismiss that statement as mere nonsense, because it was written 740 years ago or so, will not quite do, as anyone, I think, will agree who will carefully read the *Itinerarium* in its entirety. The man who wrote it was a good deal of a realist, with, furthermore, a pretty taste for biological details. That he had some sort of observational basis for his remarks about the effects of continence, the truth of which he so emphatically alleges, seems highly probable. It is possible that the situation was essentially similar to that ritual chastity of the warriors of certain North American Indian tribes preparatory to an expedition, which has been particularly discussed by Frazer[14] (iii, pp. 160-161) and Briffault[15] (iii, p. 355). In addition to the most absolute chastity they starved themselves, went without sleep, lacerated themselves with briars and knives, and sometimes even

[12] The original text of the portion italicized reads as follows: "unde et audientibus iis etiam qui interfuerant, audenter protestamur, in illa peregrinatione Christianorum centum millia fuisse mortuos, pro eo tantum quod sub divinae spe mercedis se continuerunt a mulieribus ducentes iniquum jactura pudicitiae carnis obtinere sospitatem. Opposuerunt patientiam etiam usque ad corruptionem carnis, ut perseveraret impolluta castitas mentis."

[13] There would seem to be an interesting and fruitful problem here for some young and enthusiastic student of medical history. Possibly the field has already been tilled, but if so the reference has not come to my attention.

[14] Frazer, J. G. The Golden Bough. A Study in Magic and Religion. Third Edit. Part II. Taboo and the Perils of the Soul. *London* (Macmillan), 1911.

[15] Briffault, R. The Mothers. A Study of the Origins of Sentiments and Institutions. Three Vols. *New York* (Macmillan), 1929. Briffault's discussion of this particular point appears to be taken bodily from Frazer (*loc. cit.*) though he gives additional references to Dorsey's *Omaha Sociology*, which I have been wholly unable to find pertinent upon examination of the original.

mutilated themselves by cutting off fingers. The result was that they worked themselves into a delirious state, saw visions, and so on. Frazer (*loc. cit.*) says: "It is hard to conceive any course of training which could more effectually incapacitate men from the business of war than that which these foolish Indians actually adopted." It seems probable that there would have been on Richard's crusade, as indeed on any and all others, a great many extreme religious fanatics, who would zealously deny and scourge the flesh for the sake of what they were pleased to regard as their "souls," in every possible way that they could conceive of, *including* sexual abstinence. It is quite conceivable that the combined effect of all these salvational activities may have been so deleterious to health, when added to bad sanitary conditions and poor and scanty food, as to have led to an excessive mortality. On this hypothesis it is only necessary to suppose that Richard the Canon attached a somewhat greater relative importance to the sexual element in the picture than a modern health officer perhaps would, and his statement becomes credible in principle, if not in respect of the precise round number of 100,000.

One further point in this connection is perhaps worth mentioning. Richard the Canon's implied liberal, not to say modernistic, views on the salubrity of the congress of the sexes may possibly be connected with the fact that wives were not forbidden to the English priesthood until so late as 1102,[16] and there was by no means unanimity of opinion among ecclesiastical authorities at that time as to the wisdom of forbidding such unions. Henry of Huntingdon felt it to be "striving after a purity too great for human strength." And the whole history of sacerdotal celibacy indicates that, as in other times and other matters, prohibition was commonly followed by bootlegging. The present connection is that it is probable that Richard the Canon was, in his own lifetime, familiar with marriage among the clergy and possibly shared the views of many another cleric of the time that it was a healthy state.

[16] Coulton, G. G. *Art.* Celibacy. *Encyc. Brit.*, 11th Edit., Vol. 5, p. 602.

RAYMOND PEARL

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

I. EVOLUTION AND GENETICS

BAUR, ERWIN, EUGEN FISCHER, and FRITZ LENZ. Human Heredity. Translated by Eden and Cedar Paul. *New York* (The Macmillan Co.), 1931. Pp. 734. 6 × 9¼ inches. $8.00. [Bibliography of 53 titles.]

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